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PRIMARY ELASTOLYSIS

REPORT OF A CASE OF CUTIS LAXA WITH EMPHYSEMA AND A DISCUSSION OF SOME SYNDROMES CHARACTERIZED BY ELASTOLYSIS

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The term cutis laxa is used to describe a state, encountered in a variety of diseases and syndromes, where there is a loosening and stretching of the skin so that it hangs down in flaccid folds, pleats or bags. The phenomenon may be localized or generalized and may affect most or all of the integument. Cases exhibiting the state of cutis laxa are also described in the literature under a variety of headings such as loose skin, lax skin, schlaffhaut, dermatolysis, dermatochalasis, dermatomegaly, pachydermatocele, chalazoderma, chalodermie, cutis pendula, cutis pensilis and, doubtless, others. The use of cutis laxa as a synonym for cutis hyperelastica (Ehlers-Danlos syndrome) is unwarranted, but persists in spite of protest,[1] and we do not include within the scope of the term cases of pseudoxanthoma elasticum with folds of redundant skin or cases with patches of loose and atrophic skin covering some tumour, such as a haemangioma, or following some infective dermatosis.

The condition oftenest recorded under the title of cutis laxa is neurofibromatosis (von Recklinghausen's disease).

Fig. 1. Cutis laxa due to neurofibromatosis. Alibert's case from his *Monographie des Dermatoses*, 1832.

In some cases, apart from the typical lesions, tumour-like masses of connective tissue distend the skin and drag it down into pendulous bags. In other cases, large areas of abnormal skin and subcutaneous tissue hang in great thick folds from the head and neck or some part of the trunk (Fig. 1). Much rarer, and of far greater interest, are cases in which loosening of the skin, usually over most of the body surface, resembles, at first, that seen in the very aged. The sagging may, in time, proceed much further, to produce a state in which the patient resembles a wax model that is disintegrating in a fire (Fig. 2). This type of cutis laxa, often called chalodermie (von Kétly)[2] in the literature, may be present at birth or develop soon afterwards, or it may

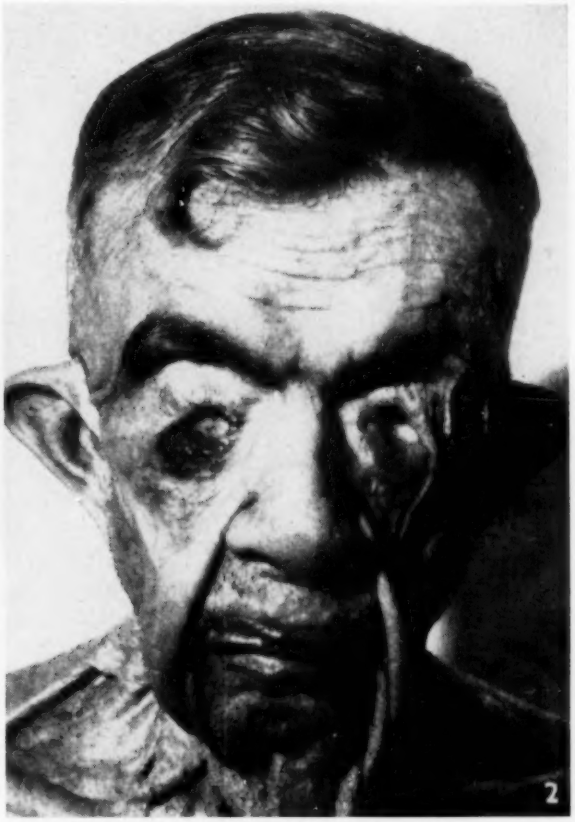
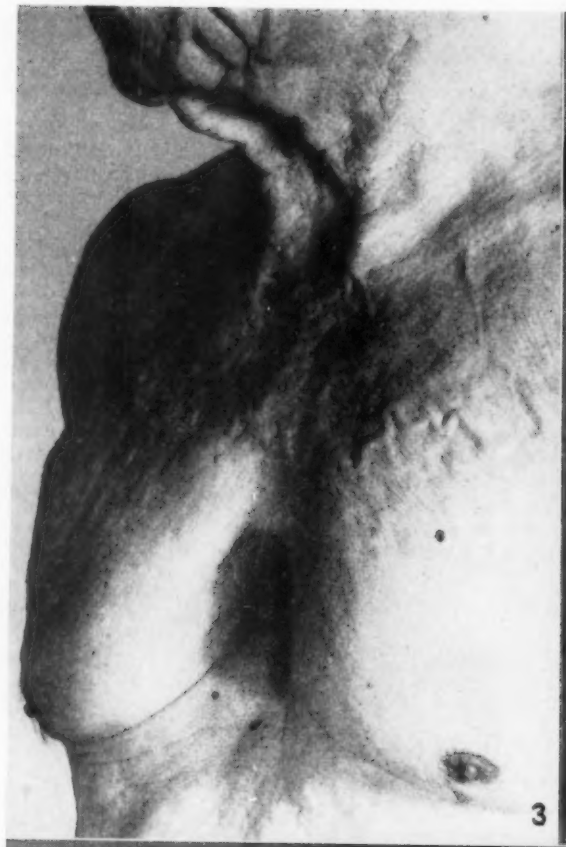
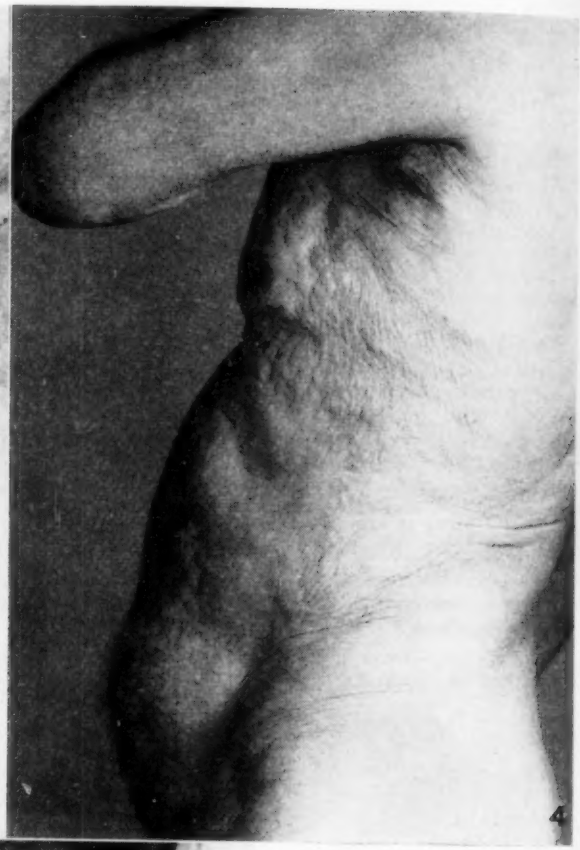


Fig. 2. Cutis laxa (chalodermie). A. G. Bettman's case.[13]





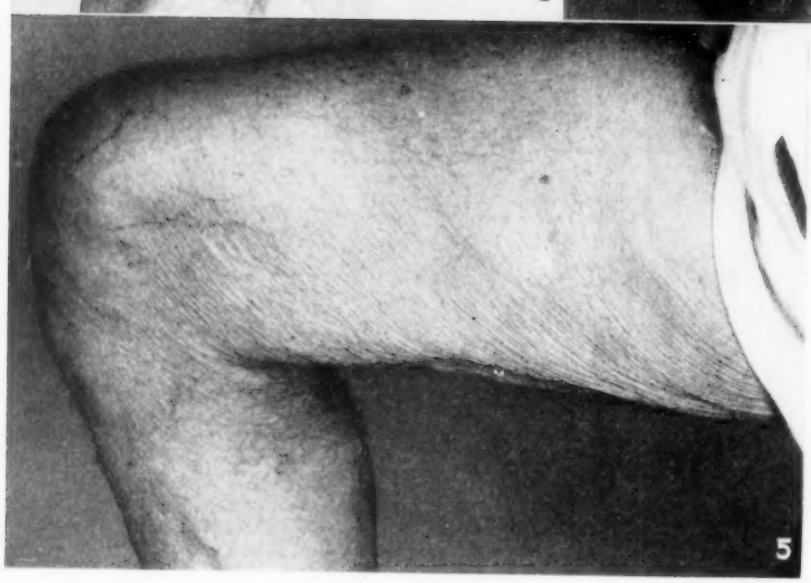


Fig. 3. Cutis laxa (chalodermie). Note prominent veins and patch of lichenified skin over sternum.

Fig. 4. Cutis laxa (chalodermie).

Fig. 5. Cutis laxa (chalodermie).

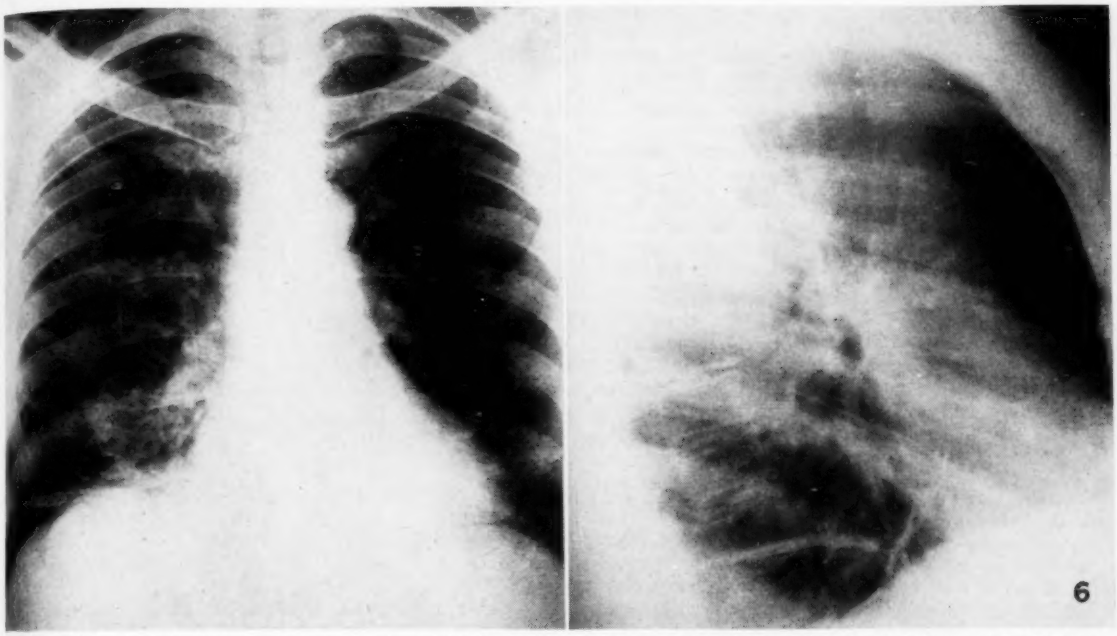


Fig. 6. Cutis laxa (chalodermie) and emphysema. Anterior and right lateral skiagram showing hypertranslucency and abnormal shadowing at the right base.

appear in childhood or adult life either after some preceding acute cutaneous or systemic disease or without any prodromal signs. The histological picture in the skin is characterized by degenerative changes in the connective tissue, particularly the elastic fibres, which are greatly diminished in quantity, and may even have disappeared entirely in some sections. Elastolysis has, in rare cases, been found to occur in organs other than the skin; after the skin, the lungs are oftenest affected with consequent emphysema, but in one known case the connective tissue of all the organs was affected.

The case we describe is one in which cutis laxa with elastolysis was associated with emphysema. The discovery of yet another case in which inelasticity of the skin is accompanied by inelasticity of the lungs goes to confirm earlier opinions that here we are dealing with a new systemic disorder of the connective tissue. We later discuss a group of diseases of the connective tissue in which elastolysis is the most prominent histological feature and classify them, for convenience and without implying that this is the only change in the connective tissue, under the title of primary elastolysis. Under this scheme of classification the present case would be one of acquired generalized cutaneous elastolysis with systemic involvement.

CASE HISTORY

The patient, a White man aged 59 years, was born in Holland and had lived in South Africa for 30 years. He was referred to one of us (J.M.) because of a patch of chronic eczema on the presternal skin which had been present for 15 years. The clinical appearance suggested a lichenified patch of seborrhoeic dermatitis and this diagnosis was confirmed by the histological changes in a biopsy specimen. It was noted on examination that the skin as a whole was excessively wrinkled, and the patient stated that this change had appeared some 3 years before and was progressive; he said he thought he had come to look 10 years older. Over about the same period he had developed a progressively increasing dyspnoea and loss of libido.

The skin of the central part of the face and scalp was normal, but elsewhere it appeared atrophic and hung in little wrinkles. A general sagging was obvious about the neck and jowls, axillae and thighs. The veins stood out prominently on the chest (Figs. 3, 4 and 5). The colour of the skin was pale, but its texture was normal, and the sweat and sebaceous glands seemed to be functioning. A fold of skin pulled out from the surface, only slowly crept back into place; there was no snap as in cutis hyperelastica. The joints could not be hyperextended.

Shortness of breath on exertion had become slowly progressive over 2 years, resulting in considerable disability since a few steps uphill or the slightest attempt to hurry on the flat caused dyspnoea. However, respiratory discomfort was absent at rest, only 2 pillows were used at night and paroxysmal noctural dyspnoea had never been experienced. He did not cough or wheeze and gave no history of chronic cough, asthma, pneumonia, angina, chest pain or palpitations. He gave up smoking 7 years previously chiefly to help cure a benign gastric ulcer.

On examination, cyanosis, clubbing and signs of cardiac failure were absent. His height was 6 feet and his weight 185 lb. Although he was free from dyspnoea at rest it became very noticeable after climbing one flight of stairs. The somewhat barrel-shaped chest had a circumference of 41 inches on expiration, expanding to 43 inches on full inspiration. There was no kyphosco'iosis or chest deformity. The large tortuous superficial veins on the left upper arm and chest did not show reversed blood flow, and their prominence seemed to be due to loss of tissue support. The jugular venous pressure and pulsations were normal. The lung fields were hyperresonant, air-entry appeared equal and normal, tactile vocal fremitus was slightly diminished, and crepitations and rhonchi were absent.

The heart sounds were distant, but triple rhythm and murmurs were absent. The blood pressure was 110/60 mm. Hg. Peripheral arterial pulses were all palpable and normal. Ophthalmoscopy revealed no angioid streaks or other abnormalities. The urine contained no protein, glucose, bile pigment or urobilin. The genitalia showed no abnormality; liver cirrhosis and gynaecomastia were absent, but further investigations to determine the nature of his impotence were not permitted. No abnormalities

were detected in the central nervous, gastro-intestinal and haemopoietic systems.

The electrocardiogram revealed a sinus rhythm of 68 per minute, P-R and QRS intervals 0·20 and 0·09 second, respectively, left axis deviation with clockwise rotation, the transition zone being at V5. There was no right atrial or ventricular hypertrophy. The graph was considered within normal range.

The skiagram (Fig. 6) showed a normal cardiac shadow and no aortic dilatation. There was abnormal linear shadowing at the right base with hypertranslucency below, especially striking on screening during deep inspiration. The appearances suggested emphysema, but no lung cysts or bullae were detectable.

Respiratory function tests showed moderate obstructive ventilatory defect with slight anoxaemia and hypercapnia (Table I).

TABLE I. RESPIRATORY FUNCTION TESTS

	Observed	% *Normal*	*Normal range*
Vital capacity (ml.) ..	4,150	108%	3,840
Timed vital capacity (ml.)			
1 sec. % of total ..	42%		>80%
2 sec. % of total ..	54%		
Maximum breathing capacity (litre/min.) ..	52	45%	114

Spirogram tracing showed slight air trapping and moderate prolongation of expiration.

BLOOD STUDIES

		Measured value	*Normal*
Haemoglobin		15·7 g.%	
O_2 capacity		21 vols.%	
Arterial oxygen saturation (% saturation)			
Van Slyke	At rest	91%	94 - 98
	Breathing 100% O_2 $7\frac{1}{2}$ mins.	103%	100
Oximeter	After exercise ..	77%	90 - 94·2
Arterial CO_2 content (vol.%) ..		57·5	49

The anoxaemia was corrected by breathing 100% oxygen, but after exercise the oxygen saturation dropped to 77%. The maximum breathing capacity was only 45% of normal and the timed vital capacity was abnormal.

A blood-cell count showed no deviation from the normal. Serum proteins: Albumin 5 g.% and globulin 1·4 g.%, with normal distribution of fractions. Urea 28 mg.%. Serum cholesterol 352 mg.%.

Histopathology

Two biopsy specimens were taken, the first from the eczematous patch over the sternum and the second from lax but otherwise normal skin on the left breast near the anterior axillary line.

Sections of the first specimen show a typical chronic eczematous lesion with spongiosis of the epidermis and slight parakeratosis (Fig. 7). There is a chronic inflammatory infiltrate in the upper third of the corium. Only a few elastic fibres are to be seen in the whole of the corium and in the walls of the blood vessels. The collagen fibres in the deeper layers of the corium are somewhat thickened. The epidermal appendages are normal.

The second biopsy shows no eczema (Figs. 8 and 9). The epidermis is slightly atrophic with a few short rete pegs. Verhoeff and orcein stainings reveal a small number of elastic fibres, which are fragmented and very short and display various stages of degeneration. The ends of the elastic fibres are thickened and bulbous or spirally twisted. The same changes are seen in the elastic of the blood vessel walls. In a few areas there are some small perivascular infiltrates consisting of lymphocytes and histiocytes. There is no evidence of previous inflammation except for slight thickening of collagen fibres in the depths of the corium; collagen fibres near the epidermis are thinner than normal. Sebaceous and sweat glands are normal.

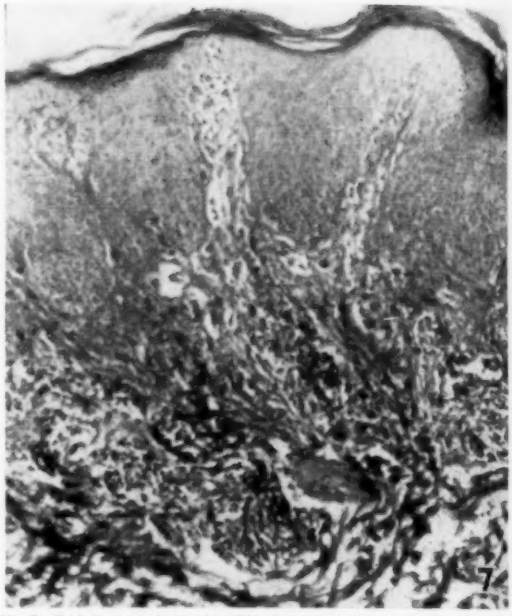


Fig. 7. Cutis laxa (chalodermie). Skin from eczematous area showing parakeratosis, spongiosis and dermal infiltrate. Only a few elastic fibres can be seen at left centre. (Verhoeff: low power.)

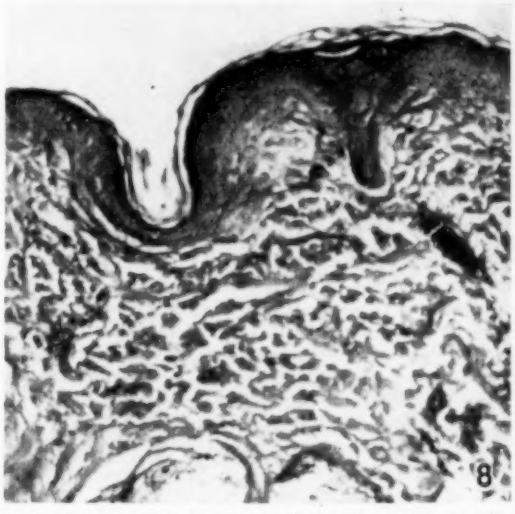


Fig. 8. Cutis laxa (chalodermie). Skin from axillary region. No inflammatory changes, but marked destruction of elastic tissue. Scanty elastic fibres at left. (Verhoeff: low power.)

The histological findings suggest a primary degeneration of the connective tissue which mainly concerns the elastic fibres. The destruction of elastic fibres is not due to any inflammatory process; if this were so we should expect to find signs of a previous or a chronic inflammation such as hyalinosis of collagen fibres or changes in the epidermal appendages or blood vessels. The

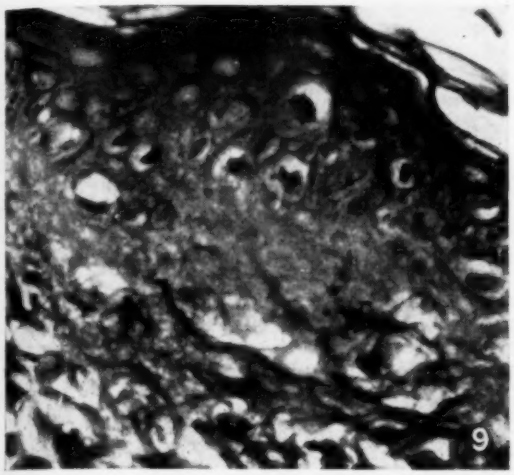


Fig. 9. Cutis laxa (chalodermie). Skin from axillary region. The black elastic fibres show marked degeneration, fragmentation and bulbous thickenings. Collagen fibres beneath the epidermis are thinner than normal. (Verhoeff: high power.)

minor, circumscribed perivascular infiltrates are not considered to be causally related to the diffuse elastolysis; they might, indeed, be related to changes in the mechanical properties of the skin.

It seems from a histological point of view that there are two different types of primary elastolysis. In one group the changes in the elastic fibres are accompanied or caused by inflammation in the skin; in the other there is no evidence of inflammation and no change to suggest what factor or agent is producing the elastolysis. Our case falls in the second group. This histological classification is in accord with that offered by Gans and Steigleder[3] in their description of chalodermie.

DISCUSSION

The clinical syndromes which, we feel, should be collected under the heading of primary elastolysis include cutis laxa (chalodermie) of the congenital and acquired types with or without evidence of systemic involvement, blepharochalasis, and the primary anetodermas (Table II). In all these conditions

TABLE II. PRIMARY ELASTOLYSIS

A. *Generalized Cutaneous Elastolysis*		
1. Congenital	(*a*) apparently purely cutaneous	
	(*b*) with systemic involvement	
2. Acquired	(*a*) apparently purely cutaneous	
	(*b*) with systemic involvement	
B. *Localized Cutaneous Elastolysis*		
1. Blepharochalasis		
2. The primary anetodermas.		

a constant feature is degeneration of the connective tissues of the skin. The collagen fibres are seldom markedly degenerate and they may, indeed, appear normal, but the elastic fibres are always reduced and fragmented and they may seem, in some biopsy specimens, to have disappeared entirely. Non-specific dermal changes sometimes noted in the early stages in cases of acquired elastolysis include dilatation or thrombosis of blood vessels, oedema, and a mild and predominantly perivascular infiltrate of lymphocytes, histiocytes, plasma cells and mast cells. The epidermis is often a little atrophic, and flattened at the line of junction with the dermis. The hair and nails and the sweat and sebaceous glands are usually quite normal.

Degeneration of elastic fibres is a common feature after healing of many inflammatory diseases of the skin. It may be marked in the scars of acrodermatitis chronica atrophicans or in those following syphilitic or tuberculous lesions, but in few diseases unmarked by gross inflammatory changes does the degeneration always, or even often, go so far as it does in cases of primary elastolysis. The changes seen in the connective tissue of senile skin (senile elastosis) and in pseudoxanthoma elasticum (systemic elastorrhexis) are in no way comparable.

The skin in cases of generalized elastolysis, whether congenital or acquired, is pallid, thin, loose and inelastic. A fold taken between finger and thumb is paper thin, unlike the thick and sometimes tumorous folds of tissue found in von Recklinghausen's disease. The texture and feel of the skin are reasonably normal; although inelastic, it is not dry and atrophic like senile skin

Atrophic changes in the gonads, with impotence in the male, are frequently described in cases of generalized cutaneous elastolysis.

Generalized Cutaneous Elastolysis

Congenital Elastolysis

The phenomenon of lax skin may be obvious at birth or appear soon afterwards, and it has even been recorded in a 6-month foetus (Houel, cited by Petges and Lecoulant[4]). Within a year or two the sagging of the skin may have progressed so far that the child, with drooping face, presents the mournful appearance of advanced old age. The facial changes are so stereotyped that affected children look almost identical.

Such a case, in a girl aged 3 years, was reported by Robinson and Ellis.[5] The skin was excessively wrinkled at birth and became progressively looser. Growth rate and mental development were normal. No defects were found apart from the skin, and there were no other such cases in the family. The dermal collagen was normal, but the elastic fibres were abnormal and reduced in quantity.

Similar cases have been described by Siemens and Eindhoven,[6] Debré *et al.*,[7] Ronchese,[8] and others are noted by Petges and Lecoulant[4] in the *Nouvelle Pratique Dermatologique* (*Dermatolysie ou cutis laxa*) which gives an excellent historical survey of the whole question.

The condition described by Petges and Lecoulant[4] as *géromorphisme cutané*, *gérodermie génitodystrophique* or *sénilisme* in the same book (*Atrophies congénitales*[4]) appears to be identical with congenital elastolysis. Affected males are impotent, with infantile genitals and the voice of a castrate; height is often less than average, and hair is scanty on the face, axillae and pubis, but normal on the scalp. Only the skin appears to be affected and sufferers may live to a ripe old age; this state is not related to progeria.

Christiaens *et al.*[9] have reported a case in a female child in which lax skin and emphysema were associated. Dyspnoea was present from birth, and the skin was seen to be abnormally wrinkled when the child was a few months old. At the age of 1 year the face looked like that of an old woman and the skin seemed too big for the body. The child died of bronchopneumonia at the age of 2 years. The dermis

was wide and had a cicatricial aspect, with large bundles of thick, short hyalinized collagen fibres; the elastic fibres were degenerate and diminished in quantity. The lungs showed both generalized emphysema and cyst formation. The authors consider the condition to be a congenital dystrophy affecting elastic tissue and compare it to pseudoxanthoma elasticum.

Bakker[10] recently reported on the case first described by Siemens and Eindhoven.[6] At the age of 18 months the child showed cutis laxa that involved all the skin and scalp. At 17 years there was a marked improvement in the state of the skin over the body and limbs; it was lax and satiny but looked fairly normal, with only a little sagging at the axillae, elbows and breasts. The face was still markedly affected and the patient looked very much older than his real age. Since earliest schooldays there had been a gradually increasing dyspnoea, which was shown to be due to severe bilateral emphysema. The author considers the condition to be due to a systemic affection of elastic tissue.

Acquired Elastolysis

Loose skin may develop in childhood or adult life, commonly without any preceding symptoms as in the classic case of von Kétly[2] or that of Carney and Nomland,[11] but occasionally after some dermatosis or febrile systemic disorder. In a case described by Goth,[12] articular rheumatism preceded the onset of skin changes in a young woman. Petges and Lecoulant[4] cite the case (of W. Dubreuihl) of a girl of 13 where fever and an eruption of urticaria-like papules were quickly succeeded by a generalized oedematous swelling of the skin and, finally, cutis laxa.

Most of the body surface, even the scalp, may be affected, and from the illustrations in the literature it would appear that the sagging of the skin is often much more pronounced in the acquired disease than in the congenital. In some cases the disease advances rapidly at first and then halts, in others it is slowly progressive.

The most interesting case of acquired generalized elastolysis is that described by Bettman[13] in a man of 62 years. The disease had begun 13 years before and the lax skin hung in enormous folds and pockets; vision was occluded by redundant skin, the fat had disappeared and the blood vessels stood out prominently. The wrists could be hyperextended, there was a large diverticulum in the floor of the mouth, and the patient was impotent. He died suddenly from haemorrhage into the left pleural cavity through a plaque of atherosclerosis in the descending aorta. At autopsy it was seen that the supporting stroma of the organs was so lax that they were deformed from their natural shape by gravity. There was bilateral bullous emphysema and loss of supporting tissue in the lungs; and there were countless diverticula in the large intestine. The testes showed atrophic changes, but the adrenal and thyroid glands were normal. In the pituitary gland the chromophobe cells had abundant cytoplasm and eosinophil cells were prominent. In the dermis the collagen appeared relatively normal, but elastic fibres were almost absent and those that remained were distorted and fragmented; elastic was reduced in the arterioles.

Localized Elastolysis

Blepharochalasis, which may be inherited as a dominant trait, deserves mention under this heading. The condition is usually evident in youth, but may first appear in adult life. The histological changes[3] are like those in other types of elastolysis, with an early phase of dermal oedema and mild cellular infiltrate and a late phase of atrophy and disappearance of elastic fibres.

Anetoderma

The primary anetodermas seem to us to be assimilable into the picture of elastolysis. As Degos[14] points out, anetoderma may begin in different ways, but ends with the same patches of atrophic skin. The original lesions are erythematous macules or papules in the Jadassohn type, pseudotumoral elevations in the Schweninger-Buzzi type, pruriginous urticarial lesions in the Pellizari type, and bullae in the Alexander type. The histological features of anetoderma[3,15,16] are strictly comparable to those of the generalized types of elastolysis with histiolymphocytic (and rarely polymorphonuclear leukocytic) infiltrations of the dermis in the early stages, and atrophy of the skin with diminution or disappearance of elastic fibres later.

The boundary between anetoderma and generalized elastolysis is hard to define; a case of anetoderma of Pellizari illustrated by Degos[14] appears nearly identical with one described by Schuppli[17] as dermatochalasis.

The primary anetodermas are nearly as rare as generalized elastolysis. Petges notes that, as with acrodermatitis chronica atrophicans, most cases have been found in Central Europe and that women are affected oftener than men. The course of primary anetoderma is unpredictable; in one of my cases (Schweninger-Buzzi type) the eruption appeared over a period of a few months and then progressed no further, but in another (Pellizari type) the first lesions were seen at the age of 9 years and are still erupting 40 years later. We have found nothing in the literature to suggest that systemic disorders may be encountered in cases of primary anetoderma.

CONCLUSIONS

The classification of the foregoing conditions under the general heading of primary elastolysis does not imply that we believe them to have a common cause. The occurrence of cases of congenital generalized elastolysis suggests the possibility of an inherited defect, but there is nothing in the literature to support this concept unless blepharochalasis is accepted as a *forme fruste* of the generalized disease.

The finding of clinical and histological evidence of inflammatory changes in the skin of some cases in the early stages of both the generalized and localized types of acquired elastolysis might imply that an infective factor is involved.

A point which suggests that there may be at least 2 distinct types of elastolysis is that in the few cases where systemic involvement has been demonstrated there has been no history or evidence of preceding inflammatory changes in the skin or elsewhere. We can make no comment on the significance of the testicular atrophy so often found in cases of generalized cutaneous elastolysis.

The discovery of systemic involvement in some cases leads us, as it did Christiaens[9] to compare primary elastolysis with systemic elastorrhexis (pseudoxanthoma elasticum). In this hereditary disease skin lesions are associated with widespread systemic involvement, affecting the eyes (angioid streaks), alimentary tract (gastro-intestinal haemorrhages), cardiovascular system (premature medial calcification of

blood vessels with coronary, peripheral and cerebral insufficiency), and perhaps certain endocrinopathies. However, in primary elastolysis the distribution of systemic involvement is different. The chief impact appears to be on the lungs. In our case the obstructive ventilatory defect was of special interest, because respiratory symptoms followed the onset of the skin changes, and there was no history of chronic bronchitis or asthma or any preceding pulmonary disease. The evidence strongly suggested that loss of elastic tissue in the lungs might be the primary fault, presumably caused by the same elastolytic process as that involved in the skin changes. The resultant obstructive ventilatory defect was similar to that found in emphysema following chronic bronchitis and asthma, where there is a secondary loss of elastic tissue.

SUMMARY

A case is described in which cutis laxa (chalodermie), emphysema and impotence developed in a middle-aged man.

The literature on cutis laxa is reviewed and it is proposed that one group of syndromes in this category might be classified under the title of primary elastolysis.

The phenomenon of elastolysis appears, in most cases, to be confined to the dermis, but in rare instances the connective tissue in other organs is involved, suggesting that primary elastolysis may sometimes be a systemic disorder.

We are indebted to Dr. Barry Kaplan, CSIR Cardiovascular-pulmonary Research Group, University of Cape Town, for the pulmonary-function tests and to the South African Council for Scientific and Industrial Research for defraying part of the expenses.

The clinical photographs were made by Mr. Robert Ellis, Department of Clinical Photography, Karl Bremer Hospital, Bellville, Cape.

Fig. 2 appeared originally in *Plastic and Reconstructive Surgery*[13] and is reproduced with the consent of Dr. A. G. Bettman and the publishers.

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ELECTROCARDIOGRAPHIC STUDIES X

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The Cerebro-cardiac Syndrome

A non-European male, aged 27 years, was admitted to the casualty department of the Karl Bremer Hospital on 10 October 1959. He had been assaulted some hours before his admission.

The assault took the form of a heavy blow on his head with a blunt instrument of unknown shape and size. It caused an open wound of his scalp and subsequently the patient noticed a weakness of his right arm, face and leg. He had not lost consciousness, but had been dazed. He was unaware of having been injured elsewhere. He had been quite well up to the time of his injury.

On examination he was found to have an open wound on the left temporoparietal region of his head. Neurological examination revealed a right-sided hemiparesis with diminished tone in the limbs. There was no plantar response to stimulation of the right sole. Nystagmus was present and there was involvement of cranial nerves VI and VII on the right. Pulse rate on admission was 84 per minute with a regular rhythm, and blood pressure was 130/85 mm.Hg. No other abnormal findings were present.

Special Investigations

Haemoglobin 12 g. per 100 ml., white cells 10,900 per c.mm., polymorphs 57%, lymphocytes 40%, and monocytes 3%.

X-rays of the skull showed, on the left, a fracture extending from the sphenoid into the parietal region.

An air encephalogram carried out 3 days after admission showed no abnormalities.

ELECTROCARDIOGRAM

During the course of the first night in hospital the patient developed a very rapid and irregular heart action. An electrocardiogram showed the presence of a gross ventricular dysrhythmia (Fig. 1a) with runs of ventricular tachycardia (Fig. 1b). Impulses normally conducted from the sinus node showed a right bundle-branch block pattern.

The administration of procaine amide (pronestyl) intravenously soon resulted in the resumption of a normal sinus rhythm. The electrocardiogram, however, still showed a right bundle-branch block pattern. Electrocardiograms taken on 12 October 1959, 23 October 1959, and 26 October 1959 showed

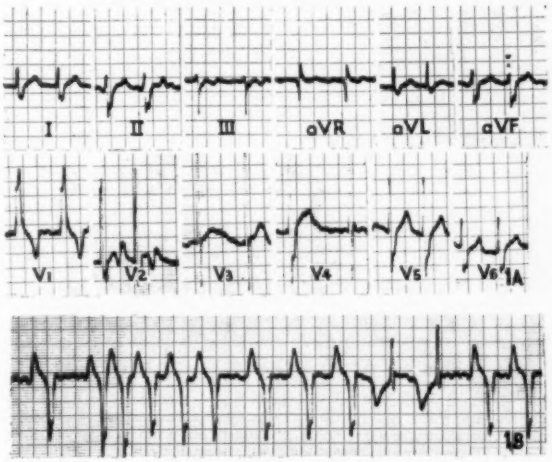

Fig. 1 (a) and (b).

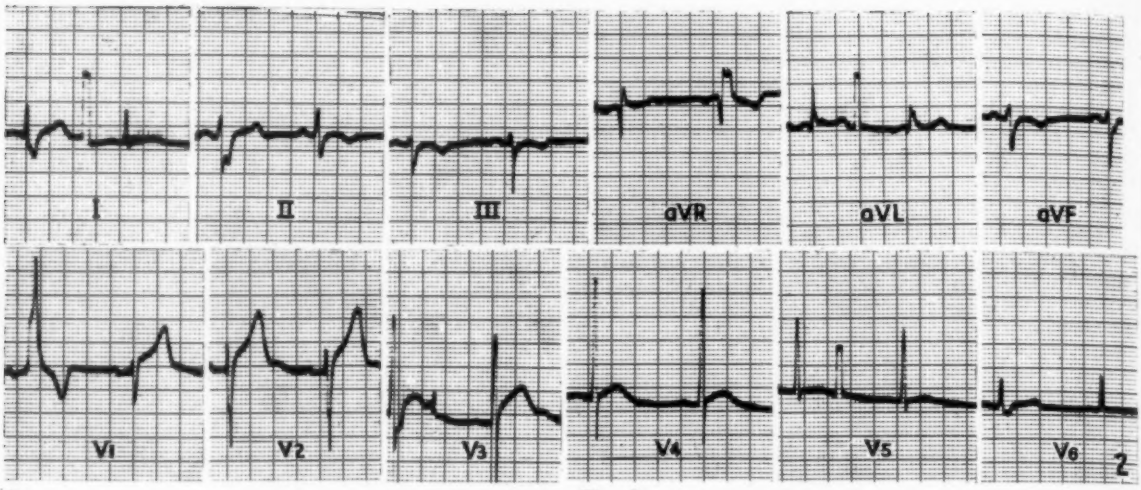

Fig. 2.

a persistence of the right bundle-branch block pattern in all complexes. Those taken on 11 November 1959 and 24 November 1959, which was the last time the patient was seen, were normal with occasional impulses still showing a right bundle-branch block pattern (Fig. 2).

The patient himself, at the time of the last electrocardiographic recording, appeared to have made a full recovery.

DISCUSSION

After having sustained a severe head injury with a fractured skull on the left side and a right-sided hemiplegia, the patient developed a serious ventricular dysrhythmia with a right bundle-branch block.

Although an indirect heart injury cannot be excluded with absolute certainty, there was no reason to suspect that this had happened. It is probable that the disturbance in rhythm was the result of the cerebral injury.

Electrocardiographic changes are known to occur with cerebrovascular accidents[1,2] and in closed head injuries. In this patient a serious disturbance in ventricular rhythm with a right bundle-branch block pattern appears to have been the result of damage to the brain. The mechanism of a disturbance of this nature is unknown; however, it may be related to the development of subendocardial haemorrhages.

OPSOMMING

'n Jong nie-Blanke manlike pasiënt het 'n ernstige hoofbesering opgedoen met 'n skedelfraktuur in die linker-temperopariëtale gebied. Daar was ook 'n regsydige hemiparese. 'n Paar uur na die besering het hy ook 'n ernstige ventrikulêre disritme met 'n beeld van regter-bondeltakblok daarby ontwikkel. Die ritmestoornis is met prokaïenamied herstel, maar die regter-bondeltakblok het 'n aantal weke lank voortgeduur.

Hierdie kardiale afwykings wat gevolg het op die breinbesering word as 'n serebrokardiale sindroom beskou.

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FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

Fifth International Congress on Nutrition. Nutrition scientists from all over the world will participate in the Fifth International Congress on Nutrition to be held in Washington, D.C., 1 - 7 September, 1960. An all-day symposium on 'World food needs and food resources' will be one of the main features of the scientific programme. The remainder of the programme will consist of 7 half-day panel discussions by invited participants, and special sessions of 10-minute papers reporting unpublished original research. Major problems of nutrition throughout the world will be reported and discussed during the meetings.

It is anticipated that some 2,500 nutritionists representing almost every country in the world will participate in the Congress. Simultaneous translation into English, Spanish, French and German will be provided at scientific sessions. A full social programme is being arranged for all participants, including special entertainment for the ladies.

Dr. C. Glen King, Executive Director of the Nutrition Foundation and President of the Congress, will open the meetings in Plenary Session on 1 September with senior United States Government officials as guests of honour. The Congress will close with an all-day symposium on 6 September, followed by an all-Congress banquet.

The programme for the symposium will be: 'The world's increasing population — major nutrition problems today', 'Today's food and population problems — views and programmes of FAO', 'Water resources of the world — food and protection of public health', 'Prospective world production and distribution of food', 'Socio-economic factors that limit needed production and consumption', 'The prospect of meeting protein needs', and 'Programme of the World Health Organization and plans for the future'.

Panel sessions are: 'Evaluation of nutritional status in man', 'Effects of processing and additives on foods', 'Lipids in health and disease', 'Animal nutrition and food production', 'Nutrition in maternal and infant feeding', 'Proteins and amino-acids in nutrition', and 'Three hours around the world — new possibilities in nutrition research'.

Prof. J. F. Brock and Dr. B. Bronte-Stewart, of the University of Cape Town, will present papers in the panel sessions on 'Dietary proteins in relation to Man's health' and 'Lipids and atherosclerosis'.

EDITORIAL : VAN DIE REDAKSIE

DR. J. J. DU PRÉ LE ROUX: AN APPRECIATION

The health service of our country has suffered a great loss in the recent retirement of Dr. J. J. du Pré le Roux from the position of Secretary for Health and Chief Health Officer for the Union. His brilliant work in this post, which he held for over eight years, is reflected in the sound and substantial advances that have been made in the health services of the Union, and his guiding personality at the head will now be greatly missed. Nevertheless the beneficient results of his work will long continue to bear fruit in the field of public health. Dr. le Roux will take a distinguished place as the fourth successor to the late Dr. J. Alexander Mitchell, the first Secretary for Health, who left an enduring monument to himself in the Public Health Act; the Food, Drugs and Disinfectants Act; and the Medical, Dental and Pharmacy Act. These statutes are the foundation on which much of our health services have been built. During Dr. le Roux's term of office important amendments have been made by the legislature to the Public Health Act; the Medical, Dental and Pharmacy Act; the Mental Disorders Act and the Nursing Act; which, with other Acts and regulations for which the Minister of Health has been responsible during this period, represent substantial extensions to the health statutes of our land.

Jonathan Johan du Pré le Roux was born at Ceres, Cape Province, on 3 August 1900. He was educated at the Boys' High School, Paarl, and studied first at the University of Cape Town and then at the University of Edinburgh, where he qualified in medicine in 1923. During 1923 and 1924 he was in private practice in England. He returned to South Africa in 1924 and, after further private practice at Hermanus, Cape, and elsewhere, he joined the Government service in 1927 as medical officer at the Westfort Leprosy Institution, Pretoria, of which, after obtaining the D.P.H. at the University of the Witwatersrand, he became Medical Superintendent in 1933.

Dr. le Roux left the Government service in 1936 on appointment as Medical Officer of Health of the Municipality of Boksburg, Transvaal, in which position he served for three years before rejoining the Union Health Department in 1939, where he was Assistant Health Officer at Cape Town and then Deputy Chief Health Officer at East London and Pretoria. It was on 1 March 1952 that he was appointed head of the Department as Secretary for Health and Chief Health Officer, from which position he retired on 3 August 1960 on attaining pensionable age.

It is noteworthy that besides having experience in private practice and in charge of a large institution Dr. le Roux held an appointment as a municipal medical officer of health. The preventive side of the health administration of this country is mainly shared between the Union Government and the local authorities, and municipal experience is most valuable in the head of the Union Department of Health, and indeed in its other health officers.

Dr. le Roux combines with profound knowledge and skill in public health, human qualities that have both endeared him to those who know him well and greatly enhanced his efficiency in his high office. His character is lofty and his personality bright and clear. He was approachable and friendly as a chief, though strict when necessary, and tactful and diplomatic in negotiations. The excellent relations that exist between the Government and the medical profession are largely the result of the confidence Dr. le Roux has inspired in the Medical Association and the Medical and Dental Council. One of the most characteristic things about him has been his personal interest in every member of his staff; on a more official level, he did a great deal in obtaining improved conditions of service for the medical personnel, with resulting increase in efficiency.

The extensions in the activity of the Union Health Department during Dr. le Roux's eight years at its head are reflected by the increase in its annual budget from £7,000,000 to £13,500,000. Perhaps the greatest achievement has been the progress made in the control of tuberculosis. The beds for tuberculosis available in the Union (including those in Government hospitals, hospitals of local authorities, mission hospitals, private hospitals, and SANTA institutions) have been increased in number from 6,000 in 1952 to 22,600 at the end of 1960.

During his eight years Dr. le Roux served as Secretary for Health under six Ministers of Health as well as one Acting Minister. These frequent changes must have made his responsibilities unduly arduous. That the Department could operate successfully in spite of them brings into relief the importance of the permanent head of department in the control of department policy. In the opinion of the Medical Association it is of the utmost importance that the headship should continue to be held by a medical man with full knowledge and experience in the problems of public health.

This account of Dr. le Roux's work is incomplete without reference to his valuable and extensive service on various councils and committees, too many, however, to enumerate here. In the past eight years he has attended meetings of the World Health Organization at Geneva and places in the continents of Africa and America on as many as nine occasions.

In placing on record this appreciation — though inadequate — of the eminent services rendered by Dr. le Roux we give expression to the esteem in which he is held by his colleagues in the medical profession.

ASPEKTE VAN GEKONTROLEERDE ASEMHALING

Selfs die onverskilligstes onder ons ondervind vroeër of later die onverbiddelike geaardheid van die Natuur se gesag. Een van hierdie wette bepaal dat ieder duim van ons sowat 2,500 vierkante duim liggaamsoppervlakte konstant 14·7 pond druk weerstaan. 'n Ander wet omskryf die samestelling van lug wat ons voortdurend en sonder

noemenswaardige verposing nodig het vir asemhaling. Diegene wat hierdie feite egter as vanselfsprekend beskou, laat twee nuwe geneeskundige vakke buite rekening, naamlik, die studie, nou reeds tien jaar oud, van menslike bestaan bokant die aarde se atmosfeer, en duikbootgeneeskunde — afgesien van die veel ouer lugvaartfisiologie. Hierdie fisiologiese oorwegings het besondere interessante en belangrike implikasies, veral ten opsigte van die moontlikheid van borskaschirurgie.

Soos Sir Robert Macintosh onlangs in Suid-Afrika kon beklemtoon, is borskaschirurgie vandag 'n triomf vir die chirurg en heeltemal binne die vermoë van byna alle pasiënte sover dit hul geestelike en liggaamlike bekwaamhede betref, hoofsaaklik weens die oplossing vir operatiewe werk in die oop borskas geskep deur intratrageale intubering met gekontroleerde asemhaling in die teenwoordigheid van relatief-geringe algemene narkose. En tog, vir miljoene is hierdie verhaal heeltemal onbekend! Russiese chirurge gebruik naamlik slegs lokale infiltrasiemetodes met behulp van niks meer nie as 0·25 tot 0·5% prokaïen, met 1 in 100,000 adrenalien. Die tegniek is onlangs noukeurig beskryf,[1] en bevestig Sir Robert se persoonlike ervarings in Rusland, China, en satelliet-lande.[2]

Op dié manier word skynbaar feitlik dieselfde gunstige resultate behaal as in die Weste, en ons het hiervóór die woord van Sir Robert Macintosh dat esofagektomie vir karsinoom sowel as oop-hartoperasies met behulp van die kunsmatige hart-long masjien beslis ingesluit moet word by hierdie statistiese prestasies. Dit laat 'n mens egter wonder oor hoeveel ongerief die pasiënt moet verduur en ook bring dit bedenkinge oor die klaarblyklike verontagsaming van die natuurlike fisiologiese oorwegings soos aangestip in ons eerste paragraaf. Hierdie verbasende Oosterse tegniek weerspieël sekerlik veel meer as bloot 'n vreemde en verskillende lewensbeskouing; inderdaad, dit is meer ‚onverskillig' as bloot ‚verskillend' of ‚anders' as ons benadering — altans so lyk dit by die eerste oogopslag. Die feit dat lokale narkose wel met welslae in Amerika vir minstens mitraalklep-valvotomie toegepas is, al gebeur dit uiters seldsaam, is geen verskoning vir 'n praktyk wat duidelik berus op 'n minderwaardige, selfs agterlike, standaard van geneeskunde nie. Van betroubare ‚statistieke' kan daar ewe-eens in hierdie verband geen sprake wees nie, al neem ons dan slegs die subjektiewe posisie van die pasiënt in ag.

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THE BLOOD VESSELS OF THE MUCOSA OF THE RECTO-SIGMOID*

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Although sigmoidoscopy has been a very common procedure for a long time, little attention has been paid to the nature of the vessels seen on the mucosal wall. A number of studies have been undertaken in order to demonstrate the effects of emotional changes on the vascularity of the mucosa of the recto-sigmoid,[1-4] changes in vascularity being measured by methods of colour comparison. But the vessels themselves have not been studied closely. Strauss,[5] a pioneer in the field of procto-sigmoidoscopy, stated: 'On the roof of the dome-shaped cavity (of the ampulla of the rectum) one often sees single narrow reddish vessels, which do not protrude above the surface of the mucous membrane, which (vessels) remind us to a certain extent of the pictures which we know from retinoscopy. Under normal conditions these vascular markings are mostly only finely indicated and are sometimes completely absent; on the other hand in pathological hyperaemias they are sometimes very prominent'.

Almy and Tulin[4] described changes in the 'veins of the mucosa' in different states of vascularity.

In performing procto-sigmoidoscopic examinations for the study of the arrangement of the blood vessels, it is essential to examine the portions of the bowel which lie ahead of the tip of the instrument, i.e. those parts which have not yet been touched by it. In this study a sigmoidoscope with distal lighting was employed, and a good telescope (6 - 8 magnification) is absolutely essential to note changes in the blood vessels. Patients were prepared by having a saline enema 4 - 5 hours before the examination, which was carried out in the knee-chest position. Anaesthetics and sedatives were never employed. Long thin (2 mm.) wooden applicators with a small piece of fine cotton wool firmly twisted round the end were used as probes.

* Based on a paper read at the Second Scientific Meeting of the Association of Physicians of South Africa (M.A.S.A.), Johannesburg, 6 - 9 July 1960.

THE VASCULAR PATTERN OF THE MUCOSA OF THE RECTO-SIGMOID

Two kinds of blood vessels are seen on endoscopy, i.e. veins and arterioles.

The Veins

The veins are most prominent in the distal portion of the rectum immediately above the anal canal. They are large vessels, blue in colour. They lie rather deep in the submucosa and appear to come closer to the surface near the anal canal. Their individual course can be easily traced, since they mostly run parallel to the longitudinal axis of the rectum. They are not much in evidence above 8 cm. from the anal margin. As they course distally they become wider in calibre. They are easily compressed and do not pulsate.

These vessels are presumably the veins of the internal haemorrhoidal plexus of the submucosa.

The Arteriolar Network

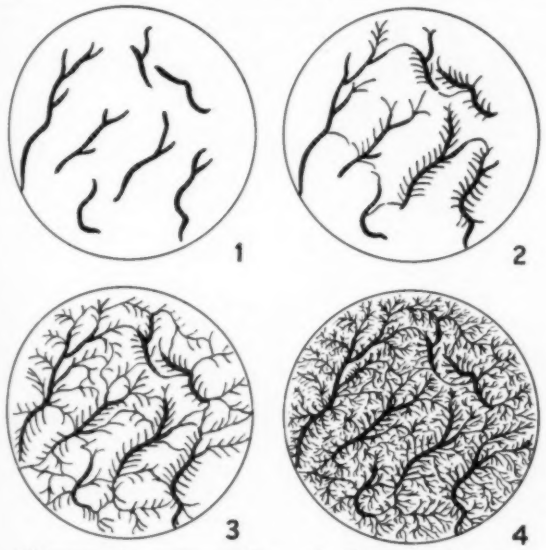
The whole surface of the mucosa is seen to be traversed by a number of small blood vessels. These vessels are very thin in calibre — under 1 millimetre — and are bright red in colour. They are visible through the surface layers of the cells of the mucous membrane which

is thin and transparent enough to make the vessels visible.

These arterioles are seen to arborize and intercommunicate to form a network or plexus. They may be seen in all stages of dilatation. In a state of vasoconstriction the finer vessels are closed off, or they are so thin that they are not visible; only the few relatively large arterioles remain open and appear as isolated vessels. In a state of vasodilatation, which can be brought about by certain stimuli to be described below, numerous tributaries open, branches anastomose with each other, and increasingly large areas of the surface are covered with fine blood vessels.

The larger arterioles lie deep to the mucosa, and when the whole arteriolar system dilates it leads to the opening up of the minute vessels on the surface of the mucosa. When these minute vessels become dilated they produce a diffuse redness of the surface, so that the deeper-lying arterioles cannot be seen through it. When the minute vessels are contracted the deeper-lying larger arterioles can be seen through the pale mucosal layer; when they are dilated, the arterioles cannot be seen. The closeness of the web produced by the minute superficial mucosal vessels thus determines the appearance of the 'background' against which the larger and deeper-lying arterioles may be seen.

The state of constriction or dilatation of the arteriolar network largely determines the appearance and the colour of the mucosa. In a state of extreme vasoconstriction the mucosa is light pink in colour; isolated arterioles, some crooked and branching, are dispersed at irregular intervals against a pale clear background. In a state of extreme vasodilatation individual vessels may not be discernible, since the whole mucosal surface is covered with numerous minute vessels and the mucosa assumes a uniform red colour. There are, of course, intermediate stages of vascular dilatation in which the mucosa of the recto-sigmoid varies from pale pink, something like that of a very pale retina, to a deep uniform red like the inside of the cheek.



Diagrammatic representation of the sigmoidoscopic appearances of the blood vessels of the arteriolar plexus on the mucosal surface of the recto-sigmoid:

Fig. 1. In extreme vasoconstriction.
Fig. 2. In slight vasodilatation.
Fig. 3. In more marked vasodilatation.
Fig. 4. In full vasodilatation.

At any stage, even in extreme vasodilatation, gentle pressure on the surface of the mucosa with the edge of the sigmoidoscope — a gentle stroking movement — will empty the small superficial vessels and reveal the arterioles lying underneath.

THE EFFECT OF PHYSICAL STIMULI ON THE MUCOSA — THE VASODILATOR RESPONSE

Light pressure applied to the mucosa leads to vasodilatation. If an area of mucous membrane in extreme vasoconstriction is lightly touched with a probe, the vessels are seen to open; many small branches become visible and a confluence of vessels occurs. The dilatation of the vessels can be seen as a steadily progressing process which takes some time to develop to its full extent. The vasodilator response is most easily observed in those cases where the initial colour of the mucous membrane was pale.

The degree of vasodilatation, the rapidity of the response, and the extent of the area over which it is spread depend on two factors:

1. The strength of the stimulus, i.e. the amount of pressure exerted, and
2. The length of time that the pressure stimulus is applied.

Firm pressure maintained for about 5 seconds leads to vasodilatation which can be seen to start about 10 seconds after the application of the pressure stimulus and which takes 2 - 3 minutes to reach the maximum; but it takes very much longer, 5 - 10 minutes, before the dilatation has visibly receded to its previous state.

If heat is concentrated on one area slow vasodilatation occurs. This can be brought about by using a lamp with a strong current and holding the instrument in such a way that the heat from the lamp is directed for a time on one spot.

When an area has received much stimulation by pressure or by heat, then not only do tributaries of the arterioles become apparent, but the larger arterioles appear wider.

THE STATE OF ARTERIOLAR DILATATION AT DIFFERENT AREAS

In a normal case the mucous membrane rarely shows a uniform degree of vascular dilatation all over, although a certain phase predominates. The most dilated portions are often found at the edges of the rectal folds (Houston's valves), with the palest areas in the recesses which lie beneath them.

In most cases the distal portion of the rectum shows the greatest degree of vasodilatation (and redness) and the mucosa is seen to become paler as the instrument is advanced proximally. In some cases this change occurs rather abruptly above the most distal rectal fold.

In certain states, especially in the irritable spastic colon, the mucosa is found to be in state of vasodilatation. In addition, in the irritable colon the bowel wall may appear not smooth but wrinkled, the mucous membrane layer being raised up into many irregular folds. In such cases the application of pressure does not lead to any visible change in the vessels of the mucosa, but there may occur a local contraction of the mucosal folds at the site of pressure.

The folds converge on the point of the probe as if to engulf it and it takes a couple of minutes before the contractions have smoothed out again.

THE EFFECTS OF THE TOPICAL APPLICATION OF DRUGS

Dilute solutions of a few drugs were dropped on the mucosa and their effects noted.

Adrenaline produced vasoconstriction which was discernible up to a dilution of 1:250,000. The effect of adrenaline was firstly to constrict the minute surface vessels, leading to a lighter 'background' against which the deeper arterioles stood out more prominently; then, as the adrenaline was absorbed, it produced a narrowing of the arterioles of the submucosa, until, with a strong solution of adrenaline, they disappeared from view entirely.

Nor-adrenaline (arterenol) produced vasoconstriction in a dilution of 1:1,000; its effect was not as marked as that of adrenaline. Cocaine (1:100) produced a slow constriction. Phentolamine (rogitine), histamine and neostigmine solutions were also applied, but the results were not constant.

DISCUSSION

The vascular response of the skin to stroking has been described in detail by Lewis.[6] He demonstrated that injuries to the skin, such as can be produced by heavy stroking, led to the liberation of a histamine-like substance (the 'H-substance') in the tissues. This 'H-substance' has 3 actions upon the blood vessels of the skin; firstly, it dilates the minute vessels locally, producing a red line limited to the area stroked; secondly, it acts on the nervous mechanism producing (reflexly) an arteriolar dilatation of the area — the flare; and thirdly it increases the permeability of the minute vessels locally which leads, in susceptible subjects, to the formation of a wheal. The whole vascular reaction of the skin constitutes the 'triple response'.

In the mucosa of the recto-sigmoid the actual process of the dilatation of the arterioles, which occurs as the result of mechanical stimulation, can be watched and studied in detail. It has been impossible to demonstrate, in the mucosa, the occurrence of a stage comparable to the local red reaction of the skin. The dilatation of the mucosal arteriolar plexus, which is seen to occur in response to mechanical stimulation in normal individuals, appears to correspond to the second stage, the flare, of the triple response of the skin. Like the flare reaction of the skin there is a latent period between the application of the pressure stimulus and the onset of dilatation, and its development and extension. The degree and rapidity of the vasodilatation response are related to the amount of the stimulus. The vasodilatation takes some time to return to its previous state. The area of visible dilatation shows a spread beyond the area which has been stimulated. All these facts suggest that a chemical substance is liberated in the mucosa which, acting on the local nervous mechanism, produces a widespread arteriolar dilatation.

SUMMARY

1. A network or plexus of arterioles can be seen by procto-sigmoidoscopic examination on the mucosal surface of the recto-sigmoid.

2. The arteriolar plexus may be in a state of constriction or dilatation. In a state of constriction the arterioles appear as discrete vessels and the colour of the mucosa is pale pink. In a state of dilatation, the arterioles branch and inter-communicate, and the colour of the mucosa becomes red.

3. In normal individuals it was found that mechanical stimulation of the mucosal surface of the bowel wall produced an area of vasodilatation.

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THE MUCOSA OF THE RECTO-SIGMOID IN ULCERATIVE COLITIS*

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In the article on 'The blood vessels of the mucosa of the recto-sigmoid' (p. 730 of this issue of the *Journal*) it has been shown that a network of arterioles can be seen on the mucosal surface of the recto-sigmoid of normal individuals and that these vessels dilate on the application of light pressure to the bowel wall. In this article the appearances of the mucosal surface of the bowel wall in cases of ulcerative colitis will be described. It was found that in ulcerative colitis subjects the application of light pressure to the bowel wall led to wheal formation, an area of localized oedema being produced.

Ulcerative colitis frequently manifests itself as a cyclical disease, periods of activity alternating with periods of quiescence — the so-called 'remitting' type. The following descriptions are based on observations made during frequent sigmoidoscopic examination of 60 patients suffering from ulcerative colitis of various grades of severity, who have been under my care during the last 6 years. Particular attention was paid to the endoscopic appearances in the quiescent stage of the remitting type of the disease during the process of healing and in the stage of remission. The examinations of some patients in the stage of a long remission and of patients with very mild ulcerative colitis limited to the rectum and recto-sigmoid provided the most useful information. The same technique was employed as in the previous investigation (p. 730 of this issue of the *Journal*).

Appearances of the Mucosa during the Healing and Quiescent Stages of the Disease

The sigmoidoscopic appearances in the acute or active stage of ulcerative colitis are well known and need not

* Based on a paper read at the Second Scientific Meeting of the Association of Physicians of South Africa (M.A.S.A.), Johannesburg, 6 - 9 July 1960.

be repeated here. When a patient begins to improve and a stage of remission is reached, the sigmoidoscopic appearances of the bowel wall are also seen to improve. Less blood and pus are visible, and the mucosal surface is less haemorrhagic and gives place to a 'granular' appearance; it is sometimes studded with bleeding points. The granules are at first large and coarse and later become much finer, while the surface may still be very friable, bleeding easily when touched. With still further improvement a stage is reached when no haemorrhages are seen; the mucosa is no longer friable and does not bleed when touched, but it is still oedematous and the surface is not smooth, but raised, and has a pink 'velvety' appearance. This velvety appearance may last for many months without the occurrence of haemorrhage. It should be emphasized that the last sign of disease to disappear is oedema of the bowel wall. As long as oedema is present the mucosal vessels cannot be visualized through the swollen mucous membrane. It is only when all the oedema has disappeared and the vessels are clearly visible that the bowel wall has returned to a normal appearance.

The process of resolution is not always uniform. In some cases the area involved steadily decreases in size, the appearances returning to normal from above downwards, so that the lowest part of the rectum is the last to heal. In other cases the healing takes place in a patchy distribution so that normal-looking areas of mucosa are interspersed with areas of oedema and haemorrhage. The recesses below Houston's valves are frequently the first areas to become normal.

The sigmoidoscopic appearances and the clinical picture do not always follow a parallel course; patients may be symptom-free and yet the rectum and sigmoid may show a considerable degree of involvement.

In a certain number of cases, especially in those with a mild localized form of the disease affecting only the distal portion of the bowel, a stage of remission may be reached when the sigmoidoscopic examination shows no haemorrhages, no friability of the mucosa, and no granularity; when all traces of oedema have disappeared and the mucosa is smooth after having regained its translucency so that the vessels of the arteriolar plexus can be seen against a clear background; when, in fact, the bowel wall appears perfectly normal. In such cases the vascular structure of the mucosa is found to be no different from that of normal individuals — there are the large prominent blue veins and the network of arterioles and minute vessels.

The Colour of the Mucosa of the Recto-sigmoid in Patients with Ulcerative Colitis

It is frequently stated that the bowel wall in ulcerative colitis is hyperaemic. This is not my experience in cases examined in the stage of complete sigmoidoscopic remission. In such cases the bowel wall is paler than in normal individuals and the vessels are contracted. Among 50 consecutive cases examined, 24 patients showed at some time in the course of their illness a stage of remission when at least certain portions of the bowel wall were seen to be normal-looking, the vessels being visible against a clear background. The colour of the mucosa of 16 of the 24 patients was pale, and the vessels were contracted in the palest portions which came under observation. This is a higher proportion than that found in normal individuals. In most cases it was found that the mucosa was darkest in the most distal portions of the bowel, becoming paler higher up.

The Effect of Mechanical Stimulation of the Mucosa in Patients with Ulcerative Colitis

When a case of ulcerative colitis is examined during a stage when the whole or a portion of the visible mucosa appears perfectly normal and the vessels are visible against a pale clear background, then the application of pressure to the bowel wall (by means of a thin cotton-wool probe) leads to the development of a localized area of oedema. The oedema response obtained in these cases may be preceded by a visible vasodilatation of the arterioles, but the preliminary vasodilator response can only be observed in cases with a slow and mild oedema response. In the majority of cases the area of the mucous membrane which has been stimulated becomes red immediately, then it swells and a raised oedematous patch is formed. There is a loss of translucency over the area of the wheal so that the mucosal vessels are no longer visible. This area of oedema usually appears to be raised to 1 - 2 mm. above the surface of the surrounding mucosa. It looks like a soft raised wheal, pink or red in colour, which extends a little beyond the area of pressure, but remains localized and sharply demarcated from the surrounding mucosa. The edge of the wheal has sharply sloping sides and the surrounding field remains pale, while the larger arterioles in the circumference of the wheal become dilated and more prominent and seem to converge on it. The surface of the wheal is glistening, and it appears sometimes smooth, but more often stippled. Gentle pressure over the wheal (stroking with the edge of the sigmoidoscope) does not lead to pallor. This is in contrast to the hyperaemia produced in normal persons by mechanical stimulation which clears on stroking to reveal the vessels again. In patients with ulcerative colitis the increased pressure exerted by stroking with the instrument leads to an extension of the area of oedema.

The wheal may go on enlarging both in height and area, for a few minutes, but it remains localized with distinct sharply-demarcated edges. The strict localization of the wheal is characteristic of the oedema response in patients suffering from ulcerative colitis and differentiates it from other appearances which may superficially resemble it.

The dispersal of an area of oedema takes place very slowly. For example, in a mild case of ulcerative colitis, during a stage when the mucosa was normal-looking, a small area of oedema, ¼-inch in diameter, was produced by light pressure. This wheal was then watched and it was noticed that after 5 minutes there was only partial dispersion of the oedema. The slow dispersion of the wheal accounts for the fact that when the usual sigmoidoscopic examination is made any oedematous patches which are present or which are produced by pressure of the instrument appear to show no regression during the period of examination.

The Promptness and the Degree of the Oedema Response

The time the wheal takes to develop may vary within relatively wide limits. In some cases the wheal develops fully in 10 - 15 seconds, while in others it is not complete

even after 3 or 6 minutes. The closer the patient is to the stage of active symptoms (he may be recovering from an attack or worsening towards an attack) the more 'sensitive' he is and the larger is the wheal produced and the more prompt the response. In many cases the reaction is so rapid that the wheal is formed by the time the stimulating probe has been removed and the telescope reset for observation.

The Production of Mucosal Haemorrhage

A strong oedema response produced by mechanical stimulation is sometimes followed by the onset of haemorrhage. The haemorrhage starts as small points or very thin lines of blood on the surface of the raised wheal. The bleeding appears to be the result of the rupture of small superficial vessels. One or several such ruptures may be seen on the surface of one wheal. There is often a latent period of a minute or two, or even 5 minutes, between the application of the pressure stimulus and the appearance of the haemorrhage. When haemorrhage in a wheal occurred it was always on the surface; no submucosal bleeding was ever seen to occur.

The Occurrence of the Mucosal Response

The mucosal-oedema response to pressure was found to occur in every patient with ulcerative colitis examined during a stage when the mucous membrane was normal-looking enough (in some areas) for the vessels to be seen. Some patients were examined during several remissions, and many cases were examined on repeated occasions during one remission. On every occasion when the vessels were visible a positive oedema response was obtained. A positive response was also obtained in those patients who had been free of all symptoms for a number of years — one patient had been symptom-free for 7 years and another for 8 years.

A large number of sigmoidoscopic examinations were carried out on normal subjects and on patients suffering from a variety of functional abdominal complaints. In only 3 cases was a localized oedema response obtained — in patients suffering from functional colonic symptoms, chiefly diarrhoea and flatulence. In all others mechanical stimulation of the bowel wall led to vasodilatation and hyperaemia. In some the mucosal surface may appear irregular and wrinkled and show an exaggerated local motor activity and an excessive secretion of mucus. Certain other changes in the mucosa may superficially resemble an oedema response, but the true oedema response has 3 characteristics: the wheal is raised about 2 mm. or more, it is strictly localized, and the vessels cannot be seen through it. With the exception of the 3 cases mentioned above, none of the cases had the typical localized prominent oedema response found in all ulcerative colitis subjects.

DISCUSSION

The phenomenon of the vasodilatation of the arteriolar plexus of the mucosa of the recto-sigmoid as the result of the application of pressure in normal individuals has been discussed in the article on p. 730 of this issue of the *Journal*. Its similarity to the flare stage of the 'triple response' of the skin has been mentioned. In patients with ulcerative colitis examined during the stage of remission when the mucosa looked normal, the development of a localized area of oedema as the result of pressure seems to correspond to the third stage of the 'triple response' — the wheal. As with the skin, the oedema response of the bowel wall appears to be caused by an increased permeability of the mucosal vessels. This is probably produced by cell injury of the bowel wall when pressure is applied to it, leading to the liberation of histamine-like substances into the mucosa. Like the wheal of the skin, the mucosal oedema response is a localized phenomenon.

The significance of the mucosal oedema response in patients with ulcerative colitis is still a matter for speculation. It is possible that it is caused by previous infection — an aftermath of the process of inflammation of the bowel wall. On the other hand, the oedema response was found to occur in 3 cases who were not ulcerative colitis subjects and who apparently had not suffered from previous inflammation of the bowel. The significance of the reaction in these 3 cases is not understood. In a case of regional chronic ulcerative colitis involving the transverse colon only, the oedema response was found to be positive in the rectum although the mucosa appeared quite normal on sigmoidoscopy. A biopsy of the rectum also proved to be normal.

The finding of the oedema response in ulcerative colitis subjects may indicate a causal relationship with the manifestations of that disease. The mucosal oedema response seems to be a phenomenon of tissue hypersensitivity, and it has been suggested that tissue hypersensitivity of the bowel wall may be an aetiological factor in ulcerative colitis (Andresen[1] and Gray and Walzer[2]). Recently Kirsner and Goldgraber,[3] in an exhaustive study of the aetiological factors in ulcerative colitis, have reviewed all the evidence and have come to the conclusion that certain clinical, laboratory and histological features of the disease appear to be in accord with a primary or secondary immunological mechanism.

The site of action of the cell injury which leads to the liberation of the vasodilator substances has still to be determined. There is a possibility that the mast cells may be implicated. Mast cells contain many powerful physiological substances, including histamine, heparin, and 5 hydroxytryptamine. In a recently reported case[4] of urticaria pigmentosa, nodules were present on the skin which, when rubbed, led to localized erythema followed by whealing and fairly rapid formation of a bulla. One such nodule was shown to be a mastocytoma, with numerous mast cells present.

McGovern and Archer,[5] on the basis of histological work, came to the conclusion that 'the pathological changes in ulcerative colitis can be summarized as being essentially congestion, oedema, and muscular spasm, and ulceration is regarded as a complication following these changes'. They regard these changes as being produced by a histamine-release phenomenon which acts on the increased number of mast cells which they found to be present in the bowel wall in cases of ulcerative colitis. My own observations have demonstrated that the initial change noticed when a case of ulcerative colitis goes into relapse is the occurrence of superficial oedema, and that the last sigmoidoscopic sign to disappear when a stage of remission is reached is also the mucosal oedema. Thus, mucosal oedema appears to be the primary lesion in

ulcerative colitis. My observations also support the view that the ulceration and the inflammatory reaction are secondary to the oedema. The evidence suggests that the oedema is a local hypersensitivity reaction, and it is possible that it is mediated by the mast cells of the bowel wall.

SUMMARY

In patients who suffered from ulcerative colitis, examined during a remission and in a stage when the whole or a portion of the visible mucosal surface of the recto-sigmoid looked normal, it was found that mechanical stimulation of the normal-looking portions of the mucosa led to the development of a localized area of oedema.

The response of the mucosal vessels of the recto-sigmoid to mechanical stimulation bears a resemblance to the 'triple response' of the skin.

The primary lesion in ulcerative colitis appears to be the development of mucosal oedema. This is interpreted as a local hypersensitivity reaction.

I wish to express my sincere thanks to Dr. Barry Kaplan for his valuable assistance. I should also like to acknowledge my indebtedness to my former secretary, Mrs. J. H. Matthews, for her unfailing help.

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FATAL SARCOIDOSIS IN A YOUNG BANTU FEMALE

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Sarcoidosis is a widespread systemic disease, which usually takes a chronic, benign course. The age when the disease first manifests itself is, in the majority of cases, between 25 and 35 years. The usual organs affected are the lungs, eyes, bones, liver and spleen. The following case shows an unusual distribution and course.

CASE REPORT

A 20-year-old Bantu female was admitted to King Edward VIII Hospital, Durban, on 22 November 1957 with 6 weeks' history of dry cough, shortness of breath, and staining of sputum. X-ray showed increased size and density of the left root (Fig. 1). A tentative diagnosis of pulmonary tuberculosis was made, and the patient transferred to King George V Hospital, where routine antituberculous treatment was begun.

On admission she was orthopnoeic and staining slightly. Her general nutritional state was good. Physical signs in the chest were those of an atelectatic lung on the left. X-ray showed complete blackout on the left with mediastinal shift to the left (Fig. 2). She was about 34 weeks pregnant.

Special examinations. Blood: Haemoglobin 9·6 g. %; eosinophils 24%. Urine: albumin +. Sputum: 5 concentrations were examined and a single acid-fast bacillus was found in one of them.

Bronchoscopy showed some blood clots in the left main bronchus, which were removed. There was an inward bulge of the wall of the left main bronchus at the branching point, from which a biopsy was taken. The pathologist reported: 'A type of granuloma suggestive of tuberculosis, but not over-typical. No malignancy found.' A subsequent biopsy specimen showed inflammatory reaction only.

At this stage, about 2 months after admission, breath sounds over the left lung began to return and an X-ray on 20 January 1958 (Fig. 3) showed a fully expanded lung. The root shadow, however, appeared to be larger. On screening, the movement of the left dome of the diaphragm was seen to be restricted. A barium swallow was normal.

Several tuberculin skin tests were all negative, and all subsequent sputum examinations were also negative.

Meanwhile, on 8 January the patient was delivered normally of a healthy male infant.

In view of these findings and the growing mediastinal mass thoracotomy was performed on 4 March. A huge, hard, lobulated mass, consisting partly of enlarged mediastinal lymph nodes, was found. The mass appeared to involve part of the ascending and descending aorta. A specimen of the tumour was taken for histology, and the chest closed. There was a stormy postoperative period, with recurrence of collapse of the left lung and congestive cardiac failure.

Unfortunately the biopsy specimen was ruined overnight by an unexpected fault in the autotechnicon, and the diagnosis remained unproved.

As the appearance of the mediastinal mass suggested malignant lymphoma, intensive deep X-ray therapy was applied for 1 week, but was then discontinued because the response did not support this diagnosis and the radiologist considered

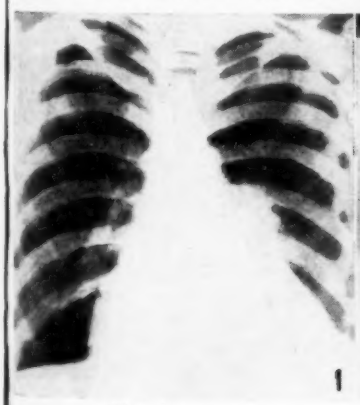


Fig. 1.

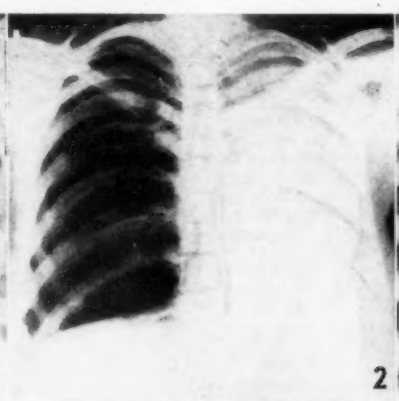
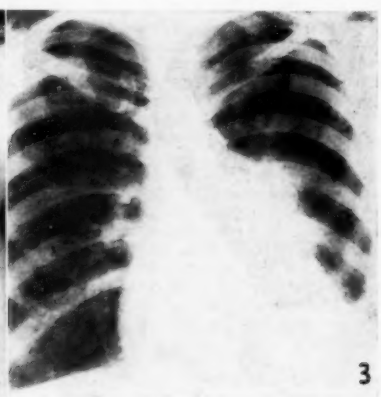


Fig. 2.



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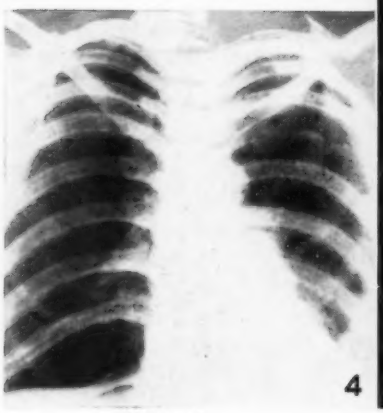
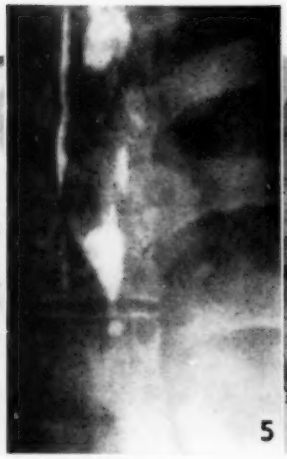


Fig. 4.



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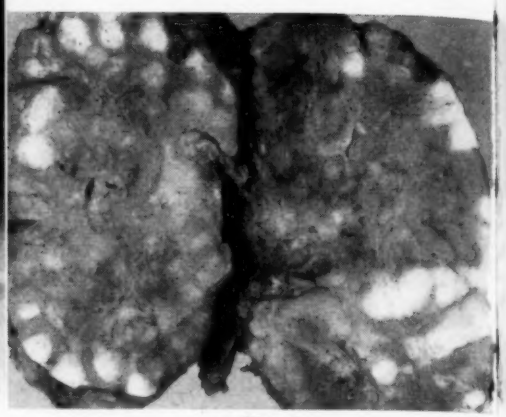

Fig. 6.

lymphatic malignancy unlikely. The patient's condition deteriorated; she complained of severe substernal pain and had two moderate frank haemoptyses. At this stage meticorten was added to the treatment.

Ten days later she complained of severe pain in the left eye, with photophobia and lacrimation. She was seen by an eye specialist, who diagnosed posterior uveitis. X-ray of skull and orbit showed no abnormality. The eye symptoms improved somewhat under treatment, but then settled into a chronic condition with exacerbations. Eventually this eye became totally blind, though other subjective eye symptoms disappeared.

In the 6th month of the illness the size of the mediastinal tumour began to diminish, and 3 months later X-rays showed further shrinkage (Fig. 4). A marked improvement took place in the patient's general condition, and she was allowed to be up and about.

By now sarcoidosis was considered an almost certain diagnosis. X-ray of the hands and feet showed small cyst-like areas of rarefaction in some of the phalanges. The test for tuberculin neutralizing factor in the serum gave negative results. The Kweim test was not done as no emulsion of sarcoid material could be obtained. Plasma proteins were normal.

The patient now seemed well, and the improvement was attributed to the meticorten therapy. She was symptom-free altogether for about 3 months, and then she started to have frequent bouts of nausea and vomiting, which could be relieved only by largactil. About 2 weeks later she complained of numbness in her right leg. On examination, motor power was found to be diminished; there was loss of superficial sensation under the knee, and loss of sense of position in the right foot; reflexes were all present and normal. Ten days later she had sudden severe pain in the region of the right sacro-iliac joint, which continued and made it difficult for her to move in bed. The X-ray report stated: 'Margins of right sacro-iloac joint not clear. This may be an infective lesion, probably TB. Area of erosion in left pubis adjacent to symphysis'. An X-ray of the chest at this stage showed further diminution of the mediastinal shadow.

The physical signs in the right leg changed considerably by November 1958, about 6 weeks after the onset of symptoms in the leg. There were now increased reflexes, with positive Babinski, ankle clonus, and wasting of the right buttock. A space-occupying intraspinal lesion was looked for. A myelogram showed a partial block at the level of D7 - 8, shown by segmentation of the myodil. (Fig. 5). CSF tests were non-committal: lymphocytes 2 - 6 per c.mm.; protein 60 - 86 mg. per 100 ml. with a slight increase of globulin; sugar 104 - 41 mg. per 100 ml.

In the 13th month after admission, small hard nodules appeared on the patient's trunk, 4 or 5 in number. Biopsy showed collagen and hyalinized fibrous tissue. About the same time the left leg became weak and the plantar responses were extensor on both sides. The patient was now completely bedridden, though she still appeared to be mentally alert.

At the end of December she started to be incontinent. On 3 January 1959 she became unconcious, though responding to stimuli. Next day she was deeply comatose, and she died the same night, 14 months after admission.

Postmortem Findings

A hard pale-coloured mediastinal tumour was found extending up to the arch of the aorta, and partly enveloping the first 4 inches of the descending portion. It extended into the hila roots on both sides. On section the tumour cut like fibrous tissue.

Both kidneys were studded with numerous small tumours of the same fibrous consistence. In fact very little normal kidney tissue remained in between these growths (Fig. 6). There were similar small hard growths in the mucosa of the stomach — the size of a pea. No such growth could be found in the liver or spleen. The skin contained the few nodules referred to above.

In the brain 2 tumours were found, and 2 in the spinal cord. Of the brain tumours, the larger, about 2·5 cm. in diameter, was situated in the white matter of the left cerebral hemisphere, adjacent to the basal nuclei. The smaller one, about 1 cm. in diameter, was adjacent to the top end of the 3rd ventricle. The 2 cord tumours, occupying nearly the whole cross-section of the cord, were found at about the levels D3 and D7 - 8 respectively.

The eyes were not examined.

Histology. Section of all these growths revealed the same picture, namely, hyalinized fibrous tissue with some remains of granulomatous tissue at the periphery — the histological picture of advanced sarcoidosis.

DISCUSSION

The diagnosis of sarcoid rests on clinical, radiological, and histological evidence. In the present case the involvement of the mediastinal glands, eye, and bones pointed to this diagnosis. The radiological picture was not typical. Only the histological examination of postmortem specimens proved the diagnosis beyond doubt.

In the differential diagnosis one had to consider, up to the time the disease was confined to the mediastinum, tuberculosis, the malignant lymphoma group, and carcinoma. Tuberculosis, apart from the negative results of all relevant investigations, seemed unlikely on the appearance of the mediastinal mass at thoracotomy. The most

likely diagnosis at that time was malignant lymphoma or carcinoma.

At the postmortem examination the mediastinal mass with the extensive spread looked like a malignant growth. The mass itself had shrunk a fair amount as compared to the appearance at thoracotomy, but not so much as appeared from the X-ray pictures. The deep X-ray therapy, in all probability, was a contributing factor to the shrinkage of the mass, as a result of fibrosis. There was also some rotation of the mediastinum, caused by the fibrosis. This rotation might account for the striking X-ray appearance of shrinkage.

The gross involvement of the kidneys was an unexpected finding at post-mortem. The patient never had any complaint regarding her kidneys and had no high blood pressure or oedema. The urine contained 1+ albumin, which in the absence of other symptoms was not considered to warrant further investigations. Sarcoidosis apparently is found, though uncommonly, to affect the kidneys. It more usually attacks the liver and spleen, but in this case these were entirely free of lesions.

The numerous sarcoid nodules found in the stomach may account for the gastric symptoms. Lesions in the mucosa of the stomach do not appear to have been previously reported.

The neurological symptoms were explained by the cord lesions found. The two deposits in the brain were silent. Although sarcoid not infrequently attacks the CNS, a number of cases having been reported, it must be quite a unique finding to have two lesions in the brain, and two in the cord, with very little abnormality of the CSF.

The age of the patient, coupled with a picture of advanced fibrosis of multiple sarcoid lesions resulting in death, makes the whole case remarkable.

SUMMARY

The case of a young girl is described who was first seen in December 1957 with collapsed left lung. The lung expanded spontaneously and a growing mediastinal mass became visible. Biopsies through bronchoscopy showed granuloma and inflammatory changes. At thoracotomy the most likely diagnosis seemed to be malignant lymphoma. Owing to a mishap biopsy material gained at thoracotomy could not be reported on. Five months later the disease started to show signs of dissemination to the left eye, bones, central nervous system, skin and stomach.

On postmortem examination there was extensive involvement of the kidneys as well as the above organs and tissues. Histology showed sarcoidosis with advanced fibrosis.

Our thanks are due to Dr. H. G. H. Houghton and Dr. M. Findlay for helpful advice. Acknowledgement is made to the Department of Health.

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THE STURGE-WEBER SYNDROME

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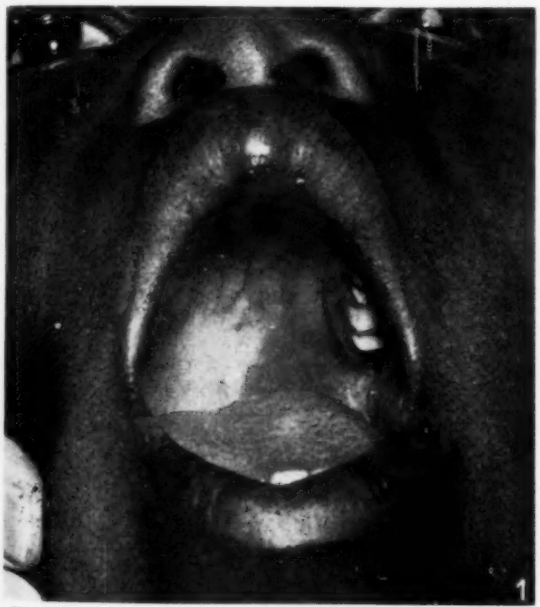

Fig. 1. Photograph of the mouth showing changes of the left side of the soft palate and left side of the tongue.

The Sturge-Weber syndrome belongs to a group of diseases known as the phakomatoses, which pathologically occupy an intermediate place between the abiotrophies and the hereditary tumours. They have in common blemishes of the skin and involvement of the central nervous system and eye. Van der Hoeve suggested the name phakomatoses. The other members of this group are:

1. Neurofibromatosis or von Recklinghausen's disease, in which there are subcutaneous nodules, tumours along nerve trunks, and pigmentary changes in the skin. Almost any part of the eye may be involved; in many instances associated developmental anomalies of the filtration angle and peripheral anterior synechia lead to buphthalmos.
2. Tuberose sclerosis or Bourneville's syndrome, in which the range of manifestations include adenoma sebaceum of the skin, with or without cerebral tumour, renal angiomata, and retinal tumour.
3. Hippel-Lindau syndrome, in which there are angiomata reticulomata of the cerebellum, spinal cord, and retina.

Historical

In 1879 Sturge[1] described a disease characterized by congenital glaucoma with coexistent flammeus and epileptiform fits of the opposite side of the body. A focal lesion of the cerebrum similar to that of the face was postulated.

In 1929 Parkes Weber[2] reported a patient with right-

sided hemiatrophy, right-sided congenital spastic hemiplegia, widespread cutaneous vascular naevi with bilateral involvement of the face, and left glaucoma. Skull X-rays revealed that the left side of the brain was more opaque than the right; no epileptic fits occurred. Later, Weber obtained radiological evidence of intracranial calcifications which he diagnosed as calcified tortuous blood vessels.

CASE REPORT

A young undernourished African boy, aged 8 years, was seen at the Alexandra Health Centre and University Clinic in December 1956. His complaint was that he had intermittent pain in his left eye and could not see out of this eye for the past 2 years. There was no history of epileptiform seizures. The only family history of a similar condition was that the mother had port-wine staining of the left shoulder.

On examination. Height 4 ft. 3 in., weight 46 lb. Blood pressure 90/50 mm.Hg. The skin was dry with some hyperkeratosis and pavementing, especially of the lower limbs. There was gross swelling of the left side of the face, port-wine staining of the left side of the hard and soft palate, the left half of the tongue (Fig. 1) and the left side of the neck reaching to the manubrium. No abnormality was found of the heart, lungs, abdomen or central nervous system.

Ocular findings. A mature cataract was present in the left eye but there was no ocular enlargement (Fig. 2). A circumcorneal flush and conjunctival injection was seen and the intra-ocular pressure was markedly raised. The pupil did not react to direct light, and light perception was absent. The patient was admitted to St. John's Ophthalmic Hospital, where his glaucoma was successfully treated with diamox and meiotics.

Fig. 2. Photograph of the left eye showing a left mature cataract with no ocular enlargement.

Fig. 3. Lateral X-ray of skull showing massive calcification of a convolutional pattern in the region of the cerebellum.

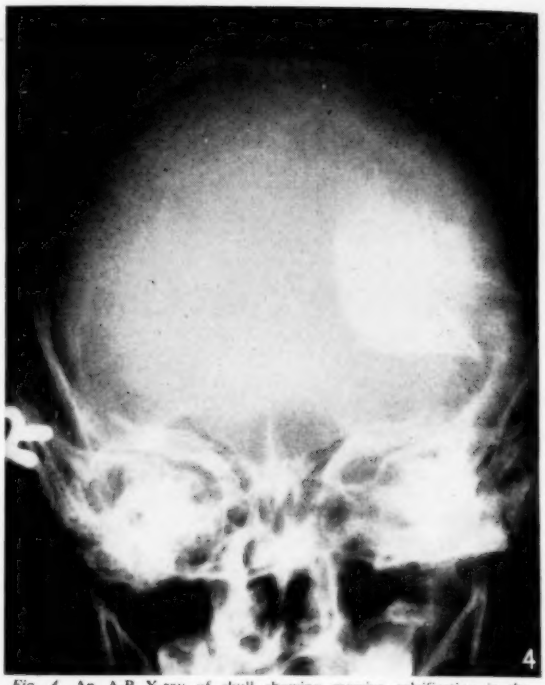

Fig. 4. An A-P X-ray of skull showing massive calcification in the left hemisphere.

X-ray. There was an extensive area of calcification presenting a convolutional pattern in the region of the cerebellum, and calcification in the left hemisphere (Figs. 3 and 4).

DISCUSSION

1. *Heredity.* The Sturge-Weber syndrome, according to Bergstrand, Olivecrona and Tönnis,[3] is due to a congenital tendency which might be inherited, but there is not such clear evidence of heredity as in the von Recklinghausen and Hippel-Lindau syndromes. In the mother of the present patient port-wine staining of the shoulder occurred but no other features of the syndrome were found.

2. *Cerebral and neural involvement.* Krabbe[4,5] demonstrated by means of biopsy that the calcifications were not confined to the pial vessels but also involved the cerebral cortex. Angiomatous changes of the pia and focal aplasia of the brain, with probable secondary sclerosis and calcification of the aplastic areas, were found. It was thought that this developmental defect was both ectodermal and mesodermal in origin. In the present case there did not appear to be any symptoms of central nervous involvement, although there was radiological evidence of extensive changes in the cerebellum.

3. *Calcifications.* Frigyer, Mattyus and Molnar[6] state that a vascular anomaly is the primary change and this explains the circumscribed nature of the clinical picture. Calcification was the most frequent sign but was not

essential to the diagnosis. The X-ray appearance of the present case was most interesting, because the calcification appeared to conform to the cerebellar convolutions of the brain and yet there were no signs of cerebellar or other neurological involvement.

4. *Ocular findings.* Duke-Elder[7] states that although glaucoma-like naevus is congenital and buphthalmic in type, cases of simple chronic glaucoma without deformation of the globe do occur. This glaucoma is often chronic and non-inflammatory, associated with deep cupped and atrophic discs, and progressing to complete blindness, though many patients may keep their sight for a long time despite increased ocular tension. In our case there was increased ocular tension but no ocular enlargement. Inigo[8] described a posterior capsular cataract in the Sturge-Weber syndrome.

SUMMARY

A case is described of the Sturge-Weber syndrome in which the interesting features are:

1. A unilateral mature cataract with glaucoma but no ocular enlargement.

2. Cerebellar calcifications without other signs or symptoms of cerebellar disturbance.

3. The possibility of hereditary transmission.

Our thanks are due to the Board of Management of the Alexandra Health Centre and University Clinic, the Superintendent of St. John's Ophthalmic Foundation, the photographic department of the University of the Witwatersrand, and the X-ray department of the Non-European Hospital, Johannesburg.

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A HEALTH SPA AFLOAT

DR. S. R. S. LAING, *Ship's Doctor, R.M.S. 'Windsor Castle'*

The importance of positive health is becoming increasingly recognized by the medical profession. This includes not only the maintenance of good health, but also regular medical examinations and the recognition of incipient disease or factors which may lead to either mental or physical illness. Many people, however, take little notice of their health until it is too late and disease strikes them down, whilst others, who would like to look after themselves properly, are often too busy or preoccupied to do so. The more progressive firms in Britain insist on their senior executives having regular full medical examinations, but such firms are more the exception than the rule.

An ideal time in which a man or woman can have a medical examination is during a sea voyage. Whilst on board there is time to spare, and consequently neither doctor nor patient need feel rushed; hence the consultation and examination can proceed as they should — leisurely, thoroughly, and in a relaxed atmosphere.

Health Centre Aboard the R.M.S. 'Windsor Castle'

It is for these reasons and to provide the busy man with an opportunity to have a thorough medical examination and a course of treatment, that the Union-Castle Mail Steamship Company's new ship, the 38,000 ton 'Windsor Castle', will have on board a health centre comprising a clinic and spa. It is expected, too, that convalescents and those suffering from diseases where treatment is ambulant and prolonged will do a complete 6 weeks voyage. From South Africa, during the winter especially, they could sail to England in 2 weeks, stay there a fortnight and return on the same ship in the care of the same doctor. Whilst in the European winter, patients could sail from Southampton to the Cape, remain there for 2 weeks or tour South Africa, joining the 'Windsor Castle' again for her homeward voyage.

It is, I believe, the first time in the history of shipping that a liner has carried a health spa. The type of patient that we expect to see will be sufferers from the more chronic diseases such as rheumatism, bronchitis, obesity, heart disease, hypertension, and 'tension states', and it is with such cases in mind that the clinic and spa have been designed.

In the clinic the aim has been to provide facilities for a full routine medical examination whilst the spa contains installations for both physiotherapy and hydrotherapy.

The Clinic

The clinic contains several additions to the equipment usually carried on board most passenger vessels. The chief of these additions are: an X-ray unit, an electrocardiograph machine, and certain laboratory facilities.

The X-ray unit will be useful not only for routine chest examination, but for the occasional accident, usually to a crew member. This will be essentially a 'screening' examination, and where abnormalities are discovered the patient will be sent to his own doctor for follow-up and for an opinion by a specialist if required.

As a matter of general interest, nearly all ships are using direct electric current and, in view of this, a portable transistor type of electrocardiograph machine is being used. The electrocardiograph machine is light, small and simple to operate, and will be used not only for the occasional cardiac emergency, which may occur, but during routine medical examinations and for the one or two heart cases we may expect to carry. Copies of the electrocardiograph will be sent to the patient's own doctor.

The most important additions to the usual laboratory investigations, such as urine analysis, which can be carried out on board ship, are ESRs, haemoglobins, white-cell counts, and prothrombin indices. Many people are on anticoagulant therapy nowadays, especially during convalescence from coronary thrombosis, and the fact that prothrombin times will be available to them should be much appreciated.

The Health Spa

This contains a physiotherapy and a hydrotherapy unit, and will be under the control of a highly trained physiotherapist.

The majority of people of middle age and over tend to suffer from various kinds of rheumatic and arthritic pains, some, of course, more than others. Many have only cursory treatment at home because they have no time for anything more prolonged, and often this has no effect because they are unable to obtain the most important therapy of all in physical medicine — rest. On a sea voyage there is abundant opportunity for both physical and mental rest, and I think that this, combined with the physiotherapy available on board, will very much help the majority of rheumatic and arthritic cases.

Special attention will also be paid to diet. Attractive weight-reducing diets will be available on board and also, where considered necessary, a low-cholesterol diet may be obtained. Every day on the menu there will be a 'health dish' designed by the chef in collaboration with the ship's surgeon, and it will also be possible to have the 'high fat, low calorie diet', if desired. A number of unsweetened mineral waters and a large number of fruit juices will be carried.

The Physiotherapy Unit

This will contain faradism, galvanism, short-wave diathermy, ultra-violet and infra-red light, a theraplinth, massage table, and wax bath. Short-wave diathermy will be particularly useful in treating osteo-arthritis and chronic infections, such as sinusitis; whilst wax baths and faradism are very helpful to patients with rheumatoid arthritis. Chronic low-back pain can frequently be much relieved by infra-red light and faradism followed by massage.

The Hydrotherapy Unit

Hydrotherapy is, I think, practised more on the European Continent than in England, and the spas of Carlsbad and Baden Baden are world renowned. The results, especially in the treatment of rheumatic conditions in the elderly, are very impressive and the patient who does not feel better after a course of treatment is rare indeed.

In the hydrotherapy unit there will be a vichy douche, an aerated bath, needle shower, and sauna bath. In the vichy douche the patient is massaged under sprays of warm water. In the aerated bath the patient lies in warm water, either fresh or salt, whilst cold air bubbles up from pipes beneath him. The needle shower consists of horizontal rings of piping, one ring above the other, in which the patient stands whilst above and below him are powerful sprays. Jets of water are thus thrown up at him from all directions and he receives a far more extensive spray than in an ordinary shower. These baths and showers not only act as general stimuli and tonics, but, in such conditions as tendinitis, capsulitis, myositis, and fibrositis, act as valuable preliminaries to massage by relaxing the skeletal musculature.

For centuries the sauna bath has been used in Finland, but it was not until after the 1939 - 1945 war that it reached many other countries, and now its popularity is fast increasing everywhere. The typical Finnish bath consists of a stone house lined with birchwood planks and with birchwood seats at various levels. A stove in one corner provides the heat, which may be as much as 215° F. On top of the stove are several large stones which not only hold the heat but provide steam, should the bathers want it, when water is thrown on them. There are birch leaf-bearing twigs in a bucket of water, and the Finns stimulate the skin by flicking themselves with these. They then run out and roll in the snow. A further bath follows, then more snow and so they alternate until they have had enough. The sauna bath, by alternately expanding and contracting the vasculature, acts as a tonic to the vascular system and assists in maintaining a healthy circulation. In particular, muscular pains are much relieved, especially if the sauna bath is followed by a massage and rest. The general tonic effect of the bath is remarkable.

The 'Windsor Castle' modifications of the Finnish sauna bath are that the stove is electrically heated instead of being heated by charcoal and wood, and long loofahs and bath brushes are supplied instead of birchwood twigs. The ship is air-conditioned throughout and there is no snow on board, but its place can be taken by one of the swimming pools, a cold needle shower, or a cold aerated bath.

The 'Mayflower' sailed from Barbican Quay, Plymouth, England, to what was later called Plymouth in Massachusetts, America, 340 years ago and she carried a barber-surgeon. We have come a long way since then! On board every ship today there is a hospital and dispensary containing drugs to a scale laid down by the government. On every passenger vessel there must be a doctor, and some of the larger vessels also carry a nursing staff; an operating theatre; an anaesthetic machine; a passenger, crew, and isolation hospital; a full set of surgical instruments; and an enormous variety of drugs.

I should like to record with much gratitude the very kind help and cooperation I have received from Dr. Arthur Levin, Chief Medical Officer of the British & Commonwealth Shipping Company, not only in the preparation of this paper but in the planning of my work on board the 'Windsor Castle'.

WORLD HEALTH ORGANIZATION

WHO OPERATION IN CONGO EMERGENCY

The World Health Organization, aided by medical teams sent by governments and national Red Cross and Red Crescent societies, is helping to contain the health situation in 5 provinces of the Congo. The first WHO doctors reached Leopoldville on 21 July. On 8 August, it was possible to announce that the provincial and many district hospitals were functioning, except for Katanga Province.

To date, 11 medical teams are deployed in the Congo, staffing hospitals and public health services, which were in many instances practically denuded of personnel, particularly at the higher professional level. Three of the teams are from the governments of Israel, Ghana and Tunisia. The other 8 are Red Cross teams from Norway, Denmark, Sweden, Finland, The Netherlands, Yugoslavia and Canada (who sent 2 teams). The Norwegian Red Cross team reached Luluabourg on 28 July. The Danish Red Cross team was deployed in Matadi on 2 August, while the Netherlands Red Cross team started work in the Blood Transfusion Centre of Leopoldville on 28 July. The 2 Canadian Red Cross teams were stationed in Coquilhatville on 4 August, while the Finnish Red Cross team is at work in the Congolese Hospital in Leopoldville. The Yugoslav Red Cross team is operating at Bukavu, as well as the Swedish Red Cross team. The Ghana Government team, consisting of 11 doctors, 4 nurses and 1 technician, is deployed in Leopoldville Province at large district hospitals, while the Israeli team, consisting of 9 doctors, 2 sanitary engineers, 1 pharmacist and 2 male nurses, is divided between Leopoldville and Stanleyville.

Greatly Reduced Personnel

Altogether, some 150 international medical personnel have arrived and are now operating in the Congo through the coordination of WHO. Under the supervision of Dr. P. M. Kaul, Assistant Director-General, and Dr. J. R. McKenzie Pollock, Senior WHO representative, WHO doctors, sanitarians and nursing advisers first made a quick survey of the health situation in order to deploy the medical teams available to the best advantage. The reports from different parts of the Congo by these experts were much the same at the earlier stages. Hospitals and public health services were still operating, but with greatly reduced personnel, particularly doctors and surgeons; nurses, on the other hand, had generally remained. In Luluabourg, for instance, out of the original 14 medical officers, only 1 surgeon and 2 physicians were found. In Kivu Province district hospitals and clinics, 8 medical officers were found to remain out of an establishment of 23. In the city of Stanleyville the total number of doctors was 12 out of an establishment of 22, while in the Oriental Province, of which Stanleyville is the capital, it appears that no doctor remained at work out of 75. In Equator Province, only 2 doctors remained out of 15 at Coquilhatville, while in district hospitals and clinics only 8 doctors, as far as could be established, remained out of 66.

No Outbreaks

These figures, although necessarily incomplete, give a picture of the situation WHO had to meet. Fanning out in the wake of the United Nations forces, medical personnel were airlifted, with the helpful cooperation of the UN Transport Control Office, into forward positions. So far, there has been no substantiated report of any serious outbreaks of disease of any kind. An outbreak of 6 cases of rabies in the Kikwit area was quickly dealt with by sending out vaccine by air.

However, there remains the long-term problem of staffing the Congolese hospitals and public health services on a permanent basis. In order to assist the Government of the Congo in tackling this difficult task, WHO has attached an advisory team of 15 staff members to the Central Ministry of Health, headed by Dr. A. Bellerive, of Haïti. There are at present no Congolese qualified doctors whatever. It is hoped, however, that a number of medical assistants, who already have undergone medical training, can be qualified as doctors within 3 - 4 years.

UNIVERSITY NEWS : UNIVERSITEITSNUUS

ASSOCIATION OF MEDICAL STUDENTS OF SOUTH AFRICA

Among the matters discussed at the Second Annual Conference of the Association of Medical Students of South Africa were the following, and it is felt that they may be of interest to the medical profession:

Mission hospitals. Interest was shown in the possibility of medical students in their clinical years being able to work at mission hospitals during vacations. It was felt that students could be of service to the hospitals, while at the same time gaining invaluable experience for themselves. The Association of Medical Students would be pleased to hear from any mission hospital which would be prepared to cooperate in this scheme and would supply details of how many students they could accommodate, for what periods they would be required and any other relevant information. The Association of Medical Students would then notify the Medical Students' Councils at the various universities who would inform the students.

General practitioners. It was also suggested that similar advantages might accrue from an arrangement whereby students, particularly in the fifth and final years, could be 'apprenticed' to general practitioners for a few weeks and accompany them on their rounds and assist in their work. The Association of Medical Students feels that, since training of this type is not provided at most South African medical schools, it could be a valuable adjunct to the training of medical students.

Would those practitioners, or hospitals, interested in the above information kindly contact either the President or Vice-President, Association of Medical Students of South Africa, c/o Students' Medical Council, Medical School, Hospital Street, Johannesburg.

PASSING EVENTS : IN DIE VERBYGAAN

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 31 August at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. T. Schrire will speak on 'Breast abscess'. All members of the Medical Association are welcome to attend this lecture.

* * *

South African Orthopaedic Association (M.A.S.A.). The Annual Congress of this Group will be held in Cape Town from Thursday 13 October to Saturday 15 October 1960. All enquiries should be addressed to the Hon. Secretary, Cape Western Sub-Group, South African Orthopaedic Association, 409 Medical Centre, Heerengracht, Cape Town; telephone 2-5161.

A full programme for the Congress will be published in the *Journal* as soon as it is available.

* * *

Suid-Afrikaanse Ortopediese Vereniging (M.V.S.A.). Die Jaarlikse Kongres van hierdie Groep sal vanaf Donderdag 13 Oktober tot Saterdag 15 Oktober te Kaapstad plaasvind. Alle navrae moet gerig word aan die Eresekretaris, Wes-Kaaplandse Sub-Groep, Suid-Afrikaanse Ortopediese Vereniging, Mediese Sentrum 409, Heerengracht, Kaapstad; telefoon 2-5161.

'n Volledige program van die Kongres sal in die *Tydskrif* gepubliseer word so gou as wat dit verkrygbaar is.

* * *

Association of Surgeons of South Africa (M.A.S.A.). The Second Biennial Congress of this Group will be held from 17 September to 20 September in Durban. The Congress will provide a full scientific programme, which will include the presentation of papers, case demonstrations and pathological demonstrations. Arrangements are being made for various social events for the entertainment of members of the Congress and their wives.

Mr. Arthur Dickson Wright, F.R.C.S., will attend the Congress as guest-of-honour. Mr. Dickson Wright is a member of the Council of the British Medical Association; Hon. Treasurer of the Imperial Cancer Research Fund; senior surgeon at St. Mary's Hospital and the Prince of Wales Hospital, London; surgical consultant to the British Railways; Vice-President of the Royal College of Surgeons, and President of the Olser Club. He is also the author of a number of well-known medical books, for example *Essentials of Medical Surgery*.

Will those practitioners who intend attending the Congress kindly inform the Hon. Secretary, Association of Surgeons of South Africa, Department of Surgery, University of Natal, Durban, if they have not already done so.

Vereniging van Chirurge van Suid-Afrika (M.V.S.A.). Die Tweede Tweejaarlikse Kongres van hierdie Groep sal van 17 tot 20 September in Durban gehou word. Op die Kongres sal 'n volledige wetenskaplike program afgehandel word wat die lewer van voordragte en die demonstrasie van gevalle en van patologiese besonderhede sal insluit. Reëlings word getref om lede van die Kongres en hul eggenotes te onthaal.

Dr. Arthur Dickson Wright, F.R.C.S., sal die Kongres bywoon as eregas. Dr. Dickson Wright is 'n lid van die Raad van die Britse Mediese Vereniging; Eresekretaris van die Imperial Cancer Research Fund; senior chirurg aan die St. Mary's-hospitaal en die Prince of Wales-hospitaal, Londen; chirurgiese konsultant vir die Britse Spoorweë; Vise-President van die Royal College of Surgeons, en President van die Olser-Klub. Hy is ook die skrywer van 'n aantal bekende mediese boeke, byvoorbeeld *Essentials of Medical Surgery*.

Die praktisyns wat van plan is om die Kongres by te woon word, indien hulle dit nie alreeds gedoen het nie, versoek om in verbinding te tree met die Eresekretaris, Vereniging van Chirurge van Suid-Afrika, Departement van Chirurgie, Universiteit van Natal, Durban.

* * *

South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-Group will be held on Tuesday 6 September at 8.15 p.m. in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape. Dr. Lorn Shore will speak on 'Allergy and asthma in childhood — comments after a recent overseas trip'. Visitors are welcome to attend this meeting.

* * *

Dr. H. Walton and Mrs. Walton (Dr. S. Wolff), of the Department of Neurology and Psychiatry, Groote Schuur Hospital, Cape Town, have left for a year's visit to New York. Dr. Walton will take up an appointment at the New York State Psychiatric Institute, and Dr. Wolff at the Child Psychiatry Unit, Creedmoor State Hospital. Dr. Walton is a recipient of a United States Public Health Postdoctoral Research Fellowship, and Dr. Wolff of a Research Fellowship from the Department of Mental Hygiene of the State of New York.

* * *

Dr. Geoffrey Dean, of Port Elizabeth, has been invited by Dr. Banyai, Chairman of the American College of Chest Physicians, to present a paper at the Sixth International Congress on Diseases of the Chest which will be held in Vienna from 28 August to 1 September 1960. The subject of his paper will be 'Air pollution and lung cancer — a South African study'. This Congress is organized by the Council on International Affairs of the American College of Chest Physicians.

Dr. and Mrs. Harold O. Hofmeyr, of Cape Town, have recently returned to the Union from a 2-months' overseas visit.

* * *

Dr. Izak Labuschagne, M.B., Ch.B. (Pret.), F.R.C.S. (Edin.), het as oor-, neus- en keelarts begin praktiseer saam met dr. W. F. Spruyt te Jennergebou 74 - 80, Jeppestraat, Johannesburg. Telefone: Spreekkamer 23-2649, woning 33-0424.

Mr. Izak Labuschagne, M.B., Ch.B. (Pret.), F.R.C.S. (Edin.), has commenced practice as an ear, nose and throat surgeon in partnership with Dr. W. F. Spruyt at 74 - 80 Jenner Chambers, Jeppe Street, Johannesburg. Telephones: Rooms 23-2649, residence 33-0424.

RECORDATI INTERNATIONAL PRIZE FOR CARDIOLOGY

The Recordati International Prize for Cardiology to the value of $2,000 will be awarded at the Fourth World Congress of Cardiology in Mexico in October 1962, for an *unpublished* paper on a cardiological subject.

Papers dealing with diagnostics, prophylaxis, and therapy will be especially welcome, but consideration will be given to any paper dealing with cardiology in the widest sense.

The papers will be judged by a jury composed of the following 5 members: Professor Chavez (Mexico City), Professor Condorelli (Rome) who will be Chairman, Professor Decourt (São Paulo), Professor Lenegre (Paris) and Professor Katz (Chicago).

Members of the medical profession of any nationality under the age of 40 may compete for the prize. In the event of a tie, preference will be given to youth. Six copies of the manuscript, typed in double spacing, of not more than 50 pages in length written in French and English and carrying not more than 10 figures suitable for the preparation of blocks of not more than 70 cm., must reach the Recordati International Prize Secretariat, Via Civitali 1, Milan, Italy, not later than 30 June 1961. Entries should be sent by registered mail. The entries must be enclosed in 2 envelopes. The outer envelope (bearing the postmarks and address) will be destroyed on arrival. The inner envelope, which must be gum-sealed, must bear no sign of any kind which might indentify the sender. The inner envelope, which will be opened personally by the Secretary, must contain: (*a*) 6 copies in each of the 2 official languages, each bearing the same motto, and (*b*) a sealed envelope bearing no mark whatsoever on the outside except the motto. This envelope shall contain a sheet giving the name, address, motto, and *curriculum vitae* of the author together with a declaration signed by him to the effect that the work has not been published.

For any further information please apply to the Secretariat, Via Civitalia 1, Milan, Italy.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

CLORPACTIN XCB

Westdene Products (Pty.) Ltd. announce the introduction of Clorpactin XCB, a new preparation designed specifically for destroying viable tumour cells in cancer surgery in order to prevent local recurrence, and supply the following information:

Clorpactin XCB is a form of monoxychlorosene with a high potency and a degree of activity approximately 4 times that of WCS-90. Gliedman *et al.*[1] found a 0·5% solution of XCB to be as effective as 10% formaldehyde in killing cancer cells, yet unlike formaldehyde it is completely safe for intraperitoneal use. Collier *et al.*[2] found XCB considerably more effective in destroying viable tumour cells than mercuric chloride, Lugol's solution, etc. Bacon *et al.*[3] state 'We . . . demonstrated repeatedly that death of carcinoma cells occurs within 3 to 5 minutes when the cells are in contact with XCB. It is for this reason that we have employed Clorpactin XCB routinely in all radical resections for cure'.

Clorpactin XCB appears to be the most effective carcinocidal agent so far discovered which can be used with safety in operative procedures. It does not harm tissue in any way, does not interfere with normal tissue growth or satisfactory wound healing, and can be used in conjunction with antibiotic or other chemotherapy. It is not incompatible with barium meals or enemas and its safety and efficacy have been confirmed by use in over 10,000 surgical procedures. There are no toxic effects, side-effects nor contra-indications when Clorpactin XCB is used in the concentrations indicated.

Clorpactin XCB is usually used as a 0·5% solution in normal saline, although, where there is a highly infected field with considerable necrosis and organic waste present, a 1% solution should be used instead. The wound must be irrigated thoroughly during the operation with 200 - 250 c.c. of solution, allowing it to remain for at least 3 minutes and preferably 5 minutes. This procedure is repeated every 30 - 45 minutes. Gloves and instruments may be dipped in the solution repeatedly throughout the operation.

Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

1. Gliedman, M. *et al.* (1958): Surg. Forum, **8**, 104.
2. Collier, R. G., McDonald, G. O. and Cole, W. H. (1959): A.M.A. Arch. Surg., **78**, 528.
3. Bacon, H. E. *et al.* (1958): J. Int. Coll. Surg., **30**, 539.

BOOK REVIEWS : BOEKBESPREKINGS

CLINICAL PHYSIOLOGY

Clinical Physiology. Edited by E. J. Moran Campbell, B.Sc., Ph.D., M.D., M.R.C.P. and C. J. Dickinson, B.A., B.Sc., B.M., M.R.C.P. Pp. xi + 530. Illustrated. English price 50s. Oxford: Blackwell Scientific Publications Ltd. 1960.

This book is a presentation by a number of physicians of the physiological concepts which they consider important for the practice of medicine. It is highly selective and rather uncritical, but it should be useful to the practising physician looking for a summary of present-day applied physiology. For the medical scientist the treatment of fundamental physiology is rather superficial. The omission of a section on the nervous system is probably wise, since current concepts in neurophysiology do not lend themselves to the very condensed method of presentation adopted.

Probably the most valuable feature of the book to the physicians for whom it is written is the section in each chapter on the rationale of diagnostic tests. Not only the simpler tests ordinarily performed by the physician himself are described but also those requiring highly specialized apparatus and laboratory technique. With the increasing tendency today to rely on such special investigations rather than on clinical observation it becomes increasingly important for the clinician to understand the significance of those investigations.

The work is sound and up to date. The rather dogmatic treatment of the subject leads to clarity of presentation. The few figures and tables are clear and helpful. Each chapter ends with a short but well-chosen list of key references. The size and the price of the book are modest and the physician who buys it will find it excellent value. A.W.S.

PHYSIOLOGY OF THE NEONATE

The Physiology of the Newborn Infant. 3rd edition. By Clement A. Smith, M.D. Pp. xii + 497. 62 figures. 95s. Oxford: Blackwell Scientific Publications Ltd. 1959.

This is a classic of paediatric literature and its encyclopaedic documentation is invaluable. The large and composite clinical summaries at the end of each chapter enable the reader to get many details as well as the general trend of the information, which is far more than the usual kind of summary provides. The index and the index of references are admirable.

The text discusses in detail the respiratory and circulatory physiology before and after birth; the changes in the blood and their relationship to icterus neonatorum; general metabolism in relation to body temperature, food, minerals and vitamins; renal physiology; neonatal endocrinology; and immunology. For those who wish merely enough documented information to support them in clinical practice, the diagrams and summaries will give complete satisfaction. For those who need more than that — physiologists, biochemists and other laboratory workers in and out of medical schools, and those interested in research — the profusion of tabulated details and references is a gold mine. An admirable book. F.J.F.

SCIENTIFIC BASIS OF MEDICINE

Lectures on the Scientific Basis of Medicine. Vol. 7, 1957-58. Pp. xii + 496. Illustrations. 45s. net. London: University of London, The Athlone Press. 1959.

This series of lectures, arranged annually by the British Postgraduate Medical Federation are designed to advance knowledge on the scientific basis of medicine. The subject matter is wide, illustrating the use of a variety of methods and disciplines by experts in their fields. Amongst the subjects covered are dye-dilution curves, the pathology of coronary-artery disease, liver-cell regeneration, and collagen structure in connective-tissue diseases. The field of genetics is discussed from a biochemical aspect and its importance illustrated by lectures on the blood groups and disorders in blood coagulation. Resistance to infection, antibody formation and the auto-antibody phenomenon are included, together with a discussion on the growth of viruses and one on poliomyelitis. Physiologists would be interested in the lectures on potassium, sensory pathways, intestinal smooth muscle, tissue-oxygen studies, the biochemistry of hypertension, and the scientific basis of otological practice. A useful book for teachers and research workers. B.B-S.

CORRESPONDENCE : BRIEWERUBRIEK

BANTOEPASIËNTE VAN PRIVATE MEDIESE PRAKTISYNS

Aan die Redakteur: Hierdie Departement se afdeling Bantoebewysburo hou 'n bevolkingsregister ten opsigte van Bantoes kragtens Wet No. 30 van 1950 en registreer ook geboortes en sterftes ten opsigte van Bantoes. Hierbenewens word ook 'n rekord gehou van vingerafdrukke en betaling van algemene belasting. Die hele Administrasie van die genoemde aspekte is op die persoonsnommer gebaseer.

Die aanwas van miljoene kaarte en die probleem van liaseerspasie en kantoorruimte kan alleen bekamp word deur rekords van afgestorwenes gereeld van die lopende rekords te verwyder. Hierdie verwydering kan alleen gedoen word nadat 'n uittreksel gemaak is uit die sterfte-aangifte. As 'n sterfte-aangif dus nie die persoonsnommer dra nie, is so 'n aangif vir die doeleindes van die Buro nutteloos.

Ten einde te probeer om persone wat met Bantoepasiënte gemoeid is te beïnvloed om persoonsnommers van afgestorwe Bantoepasiënte aan te haal op alle rekords rakende sulke afgestorwenes, is reeds skrywes gerig aan die Provinsiale Sekretarisse, sover die saak hospitale raak, en aan die Sekretaris van Gesondheid, sover die saak distriksgeneeshere raak.

Hierdie Departement is egter begerig om ook die welwillende medewerking van alle private mediese praktisyns in die Unie te verkry deur hulle te versoek om, as een van hulle Bantoepasiënte te sterwe mag kom, so 'n afgestorwene se persoonsnommer aan te haal op die geneeskundige sertifikaat van oorsaak van dood (vorm BMD 8), of op enige ander dokument wat die medikus mag teken in verband met die afsterwe van sy pasiënt. Die distriks- of assistentdistriksregistrateur van geboortes en sterftes kan dan die persoonsnommer bekom wanneer die sterfte aangemeld word.

Die mediese praktisyn neem vir sy eie rekords besonderhede van pasiënte by die eerste besoek. Indien hy aandring op die persoonsnommer en dit aanteken sal dit tot sy eie voordeel wees by die uitstuur van eise onder die ongevalle-wet en die opspoor van inligting op 'n latere geleentheid, omdat die pasiënt se identiteit dan bo alle twyfel bepaal is.

Daar is met die Noord-Transvaalse Tak van die Mediese Vereniging van Suid-Afrika geskakel, maar omdat die verskillende Takke los van mekaar funksioneer, is aan die hand gedoen dat 'n versoek in bogenoemde verband aan u gerig word vir publikasie in u *Tydskrif*. Hierdie Departement wil dus op u 'n beroep om medewerking in hierdie verband doen en verneem graag of u aan die versoek kan voldoen.

Sekretaris
Bantoe-Administrasie en -Ontwikkeling

Kantoor van die Bantoe-Administrasie en -Ontwikkeling
Posbus 384
Pretoria
6 Augustus 1960

ECTOPIA VESICAE : APPEAL FOR MATERIAL FOR INVESTIGATION

To the Editor: This Department is particularly desirous of obtaining specimens of still-births displaying ectopia vesicae and related anomalies of the infra-umbilical region of the abdominal wall. These are needed for investigation in connection with a study of the anatomy of these conditions with a view to improved surgical correction, initiated by a team of urologists in Cape Town, and now being carried on in collaboration with them by Dr. Paula Wilson, lecturer in this Department.

The literature on these anomalies, though extensive, does not provide all the information needed if operative procedures are to be devised which will avoid damaging any vital structure. Postmortem study of cases which have survived into postnatal life, whether operated or unoperated, has proved insufficient for our purpose, since the anatomy is almost invariably obscured by subsequent inflammatory processes.

In the Cape Peninsula area, this Department will gladly undertake the collection of any specimens if the doctor in charge of the case will be so good as to notify us by telephone (55-4852 : attention Mr. Coetzee).

L. H. Wells
Professor of Anatomy

Department of Anatomy
University of Cape Town
Medical School
Observatory, Cape
15 August 1960

PRESCRIBING OF COMBINATIONS OF PROPRIETARY PHARMACEUTICAL PREPARATIONS

To the Editor: With the ever-increasing volume of new proprietary pharmaceutical products appearing on the market, many containing only one active ingredient, there is a natural tendency for prescribers to wish to use a mixture of two or more liquid preparations or to prescribe the addition of other drugs to a marketed formulation in order to provide modifications in action, additional therapeutic effects, or the avoidance of side-effects. Questions on the compatibility of mixtures of certain pharmaceutical products are often referred to pharmaceutical manufacturing companies, who alone are aware of the nature of all the materials included in the formulation and of the factors governing their stability. In many cases, however, admixtures are prescribed with perhaps little more than a cursory consideration of the possibility of incompatibility, which may result in rapid loss of potency or precipitation of one or more active ingredients.

The maintenance of an optimal pH is essential for the stability of solutions of some drugs, e.g. vitamins of the B complex and certain antibiotics, and the alteration of pH by mixing the solution with other preparations or by adding a

further drug may result in rapid loss of potency, especially on storage at adverse temperatures. Changes of hydrogen-ion concentration can also result in precipitation of drugs from solution, the addition of such mildly acidic substances as alkaloidal salts serving to precipitate theophylline from many of its soluble complexes or barbiturates from solutions of their alkali metal derivatives. Changes in solvent caused by admixture can also affect the stability of liquid formulations, for instance when alcoholic solutions of one or more organic bases are diluted with a preparation of which the vehicle is aqueous.

The effect of additional aids to formulation in one preparation, e.g. stabilizing agents, solubilizing agents, preservatives, colourings, and flavourings, on the stability and potency of a second preparation are much less predictable and may even be unknown unless specific stability tests are undertaken. There would, however, be good grounds for suspecting that a reducing agent added to stabilize one product could well have a most adverse effect on the potency of the drug contained in a second product, and that solubilizing agents, whether anionic, cationic or non-ionic, could result in widespread antagonism of many drugs included in other preparations.

In view of the risks which attend the prescribing of combinations of proprietary pharmaceutical liquid preparations, it would obviously be a wise precaution to employ separate administration of two or more preparations and not to mix proprietary preparations under any circumstances.

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10 August 1960

INCOME LIMITS FOR MEDICAL AID SOCIETIES

To the Editor: Dr. N. R. Pooler[1] appears to be unaware of the fact that the preamble to the Tariff of Fees for Approved Medical Aid Societies was only agreed to by Federal Council after very full and careful consideration following protracted negotiations.

The bogy that the rules governing income limits are not being applied by societies has been raised on more than one occasion, and on each and every occasion has been successfully laid by the societies.

Whenever they have been asked to do so, the medical aid societies have proved to the satisfaction of Federal Council that these particular rules are adhered to.

With regard to Item 2 of the General Preamble 'in so far as its (the Society's) rules and regulations allow' — I can assure Dr. Pooler that this particular item led to endless discussion with representatives of the societies. Had the Medical Association not agreed to its inclusion no Tariff of Fees for Approved Medical Aid Societies would ever have been negotiated.

Accounts rendered in terms of the Tariff of Fees are usually settled promptly by medical aid societies.

Dr. Pooler reads a good deal more into the wording of Item 7 of the General Preamble than was ever intended.

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Johannesburg
9 August 1960

1. Correspondence (1960): S. Afr. Med. J., **34**, 680 (6 August).

INCOME LIMITS FOR MEDICAL AID SOCIETIES

To the Editor: Dr. N. R. Pooler's comments[1] on some of the points in the General Preamble to the Tariff of Fees for Approved Medical Aid Societies are worthy of study by the Central Committee for Contract Practice.

I should like to point out a further effect on the doctor's income due to the extension of these societies.

In 1946, less than 5% of my patients belonged to medical aid societies. I was therefore able to charge some 95% of patients a normal private fee, which was elastic, and bore some relationship to the cost of living and the cost of running a practice. In 1960 more than 80% of my patients belong to medical aid societies, leaving some 10 - 20% paying an ordinary fee. As medical aid society fees have virtually been pegged (the small increase bearing no relation whatsoever to increasing costs of practice and living), the anomalous position now is that although my practice has increased considerably, my gross income remains much the same, whereas my net income is less. This is not the law of diminishing returns and it is inconceivable that any business concern would tolerate such a state of affairs. Our profession, however, blithely accepts it and believes the myth that medical aid societies cannot afford to increase our fees. We further believe the old argument that medical aid society fees are guaranteed and payment is prompt (see Dr. Pooler's comments on this). My bad debts have increased by 1 - 1½% over the past 15 years which corresponds to the growth of the medical aid societies. Some of these bad debts are due to repudiation of the fee by societies, when the patient had been seen in what was regarded as a *bone fide* capacity. The other argument that many of these patients would attend the Government hospitals does not bear close scrutiny.

I should now like to submit the following comparative table of fees for 1939 and 1960, bearing in mind that, in 1939, the fees quoted applied to almost the whole of a doctor's practice, whereas the 1960 fees *also* now apply to almost the whole practice. The 1939 fees were agreed upon by the 4 surgeons practising in Durban. The 1960 fees are laid down in the medical aid society tariff. The only comment is that the *actual* fees received by surgeons today are, in fact *less* than those received in 1939 with the exception of the consultation fee. Does this absurd state of affairs require further comment? I pointed this out to two business men whose caustic reply was 'you should have your heads read!'

	1939			*1960*		
	£	s.	d.	£	s.	d.
Consultation fee	2	2	0	2	12	6
Operation A	15	15	0	12	10	0
Operation B	21	0	0	15	0	0
Operation C	25	0	0	20	0	0
Operation D	35	0	0	30	0	0
Operation E	42	0	0	32	10	0
Operation F	75	0	0	47	10	0

I would ask your readers to compare these figures with the cost of running a practice, of a motor-car, food, clothing etc., for the same years.

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Durban
12 August 1960

1. Correspondence (1960): S. Afr. Med. J., **34**, 680 (6 August).

ROSTER OF OBSTETRICIANS

To the Editor: Just over a year ago our organization, called Sisters Incorporated, was formed in Cape Town to aid unmarried mothers. To date we have handled 85 cases, and each week brings new patients.

In some instances the girls are from the lower income groups and are placed in various institutions and homes where their medical needs are catered for. However, we have been faced more recently with girls who come from the better-educated classes and those of a higher social standard who are able to pay for medical treatment. These girls, for obvious reasons, are unable to be placed in the established institutions, and therefore other arrangements have to be made for them. Among these is the choice of an obstetrician who is prepared to handle their cases in the strictest confidence.

Through your column we wish to ask obstetricians who may be interested to help us in this work to have their names put on our roster. Applications should be addressed to the Secretary, Sisters Incorporated, and handed personally to the Secretary of the Medical Association of South Africa, 35 Wale Street, Cape Town.

K. M. Johnston
Secretary
Sisters Incorporated
P.O. Box 95
Rondebosch, Cape
12 August 1960